The
Different Diseases of
Body

GLOSSARY.

A

ABSURD, ("The Broken Heart," act i scene 3) a scholastic term, employed when false conclusions are illogically deduced from the premises of the opponent

Affects, affections

Affronted, ("Perkin Warbeck,' act v scene 1) met face to face

Alcatote, silly fellow, a word still used in the north of Devon

Antick, ("Love's Sacrifice," act iii scene 2) antimasque (something directly opposed to the principal masque), in which grotesque and extravagant characters were introduced

B

Ban-dog, Paris-garden bandog,—a sort of mastiff kept to bait bears at Paris-garden in Southwark

Banqueting room, ("'Tis Pity she's a Whore," act v, scene 6,—stage-direction,) the room where the dessert was placed, to which after dinner, it was customary to remove

Barking church situated at the bottom of Seething Lane, was destroyed in the great fire of London

Beadsman, one bound to pray for another, a vowed servant

Beray'd, I fear my lands, and all I have, is berayed, ("Love's Sacrifice," act iv. scene 1) as those of a person condemned

Bevers, slight repasts between meals

Boar's ity, ("Perkin Warbeck," act i scene 1) an allusion to the armorial bearings of Richard the Third

Bumby, Mother, ("The Witch of Edmonton,' act iv. scene 1) a celebrated 'wise-woman', she figures in one of Lyly's plays, which is entitled *Mother Bombie*

Bonny-clabber, curds and whey, or sour butter milk

Bourd, to jest

Brach, a bitch hound

Branched velvet, ("The Witch of Ed-

(middle column)

monton," act iii scene 2) having tufts, or tassels, hanging from the shoulders

Bravery, finery of attire

Bug words, properly, terrific words, but in "Perkin Warbeck," act iii scene 2, used in the sense of high-sounding words

Bulch calf

But bolt, a strong, unbarbed arrow used in shooting at the butts

C

Casting bill a small bottle for sprinkling perfumed waters

Charm, You charm me, ('The Lover's Melancholy' act ii scene 1) you overcome my unwillingness to speak

Choppine, a large clog, worn under the shoe, and made of cork or light frame-work, covered with leather

Chouses, properly *Chiouses*, Turkish officers, rogues, swindlers

Chrisome, an infant within the first month

Cittern, } Barbers shall wear
Cittern headed } their on their cit-terns, ("The Lover's Melancholy," act ii. scene 1) A cittern headed gew-gaw, ('The I ancies, Chaste and Noble' act i scene 2), citterns, the heads of which were generally grotesquely carved, used to be kept in barbers shops for the amusement of customers

Clap dish, Stop your clap-dish, ("Love's Sacrifice', act iii scene 1) close your mouth, or hold your tongue the clap-dish was a wooden dish with a moveable cover, which was originally carried by lepers, who clapped it as a warning that alms might be given without touching them. It was after wards commonly carried by beggars

Cob-nut A cob nut out of Africa, ("The Sun's Darling," act iii scene 3) what particular fruit is here alluded to, is uncertain, cob nut is properly a large nut

Codlings, ("The Sun's Darling," act

(right column)

iii. scene 3 The Watch of Edmonton," act iii scene 1 grain us

Colts Painted colts ('The Broken Heart," act iv scene 1) rude nl silly persons, there seems t be in allusion to some allegorical picture —perhaps in the "painted cloth kind of hangings for rooms

Compass Shell keep a surer compass ("The Witch of Edmonton, act ii scene 2) an expression drawn from archery arrows shot with a certain pass, i e with a degree of elevation were supposed to go more surely to the mark

Condition, temper, disposition

Convinced, I'm sure to be convinced, ('The Broken Heart," act v scene 2) conquered, overthrown

Cot puran, one who meddles with female affairs

Counsel, In counsel, ('Fancies, Chaste and Noble act i scene 1) in secret

Courtship, the manners of behaviour necessary at court

Cousin, ("'Tis Pity she's a Whore," act ii. scene 6) nephew

Coxcomb, fool's cap

Crusted, shrivelled, roughened

Cunning, skill

Cup and knee, ('The Fancies, Chaste and Noble, act i scene 1) should probably be cap and knee'

Curst, cross, splenetic

D

Days, eight to the week, Ask any sol dier that ever received his pay but in the Low Countries, and he'll tell thee there are eight days in the week there, ('The Witch of Edmonton,' act iii scene 1) this passage is explained by the following lines of Butler,

"The soldier does it every day,
Eight to the week, for sixpence pay

Den ye, Dentlemen then ye, ("The Lady's Trial, act iv scene 2) Gentlemen, good even to ye

Desvergonzado Desver di Gonzado,

("The Lady's Trial," [...])
shame[...]; Span[...]
Devil of Edmonton, [...]
Witch of Edmon[...]
scene 1.); allusions [...]
entitled The Merry Devil [...]
ton
Dor, beetle or cockchafer
Dunstable ; I am plain Dun[...]
("The Witch of Edmonton," act [...]
scene 2.) I am blunt and honest ; in
allusion to the well-known proverb

E

Engine ; Chair with an engine. ("The
Broken Heart," stage-direction, act
iv. scene 4.) chair with moveable
arms
Engrossed, ("The Broken Heart," act
iii. scene 3.) possessed thyself of,
mastered

F

Fire i' the bed-straw, ("The Witch of
Edmonton," act iv. scene 2.) more
concealed mischief
Foist; cant term for rogue
Fond, foolish, unprofitable.
Fool ; Dressed like a fool. ("[...]
Sacrifice," act ii. scene 2. stage [...]
tion,) dressed in long petticoats,
which born fools usually wore
For ; I should have worried you, sir,
for running mad, ("'Tis Pity she's a
Whore," act i. scene 2.) to prevent
you from running mad
Forespeak, to bewitch
Fox, a cant name for a sword
Frail ; To trade by turns in such ill
frail commodities, ("The Fancies,
Chaste and Noble," act iii. scene 2.)
a play on words, frail meaning an
osier basket, in which figs &c. were
packed
Frank ; One that franks his lust in
swine-security of bestial incest,
("The Broken Heart," act iii. scene
2.) fattens ; a frank was an inclosure
in which a boar was fattened

G

Gammer Gurton, ("The Witch of Ed-
monton," act iv. scene 1.) the heroine
of the old play called Gammer Gur-
ton's Needle
Girds, taunts, sarcasms
Grogram ; As frotting as an old gro-
gram, ("Love's Sacrifice," act i.
scene 2.) an inferior kind of taffeta,
generally stiffened with gum, and
therefore liable to fret
Grammates, grammar
Guarded ; Guarded page, ("The Lo-
ver's Melancholy," act i. scene 2.) a
page with laced livery

H

Harry, harass, worry
Hench-boy, a page
Hope, ("Love's Sacrifice," act ii. scene
4., act iv. scene 2.) expect

I—J

Idle ; The youth is idle, ("The Lover's
Melancholy," act iii. scene 2.) ; I am

[...] index pointing to [...]
[...] ("The Broken Heart," [...]
[...] 1.) the index-hand [...]
[...] in the margins of old books [...]
[...] which the reader's attention was
[...] to particular passages
Ingeniously; Stand ingenious to thine
own fate, ("The Fancies, Chaste
and Noble," act i. scene 1.) [...]

[...]
Index, [...]
[...]
Itch; [...]
act ii. scene 1.) [...]
ings of wickedness and [...]

K

Ka me, kob thee, ("The Witch of Ed-
monton," act ii. scene 1.) [...]
corruption of the proverbial [...]
ka me ka thee, — do me a good turn
and I'll do you another
Know ; Not know you, ("'Tis Pity she's a
Lover's Melancholy," [...]
according to him

L

Lapwing ; Like the [...]
("The Witch of [...]
[...]
other places: there is a proverb, "The
lapwing cries tongue from heart"
Lined ; lined you, ("'Tis Pity she's a
Whore," act ii. scene 6.), pleased
Livery; Grate my hopes with any
instance of livery, ("The Broken
Heart," act iv. scene 1.), badge, orna-
ment; in allusion to the followers of
great families being distinguished by
particular badges

M

Malice ; The Lady's Trial, act iii.
scene 2.), bear malice to
Marygold, ("The Lover's Melancholy,"
act i. scene 2.), a trifle, a jest
Merchant; A ship boy is a merchant,
("The Lover's Melancholy," act ii.
scene 1.), a merchant-ship
Mew ; Mew i-absurd, ("The Broken
Heart," act i. scene 3.), a syllable-
term, when false conclusions are
drawn illogically from the premises
of the opponent
Mewed, ("The Broken Heart," act i.
scene 2.), moped [...]
its festive, a [...]
Mischief, [...] a mischief,
("'Tis Pity she's a Whore," act ii.
scene 5.), marked out to experience
some dire evil
Mockado, an inferior sort of velvet
Moon ; If the moon serve, some that
are safe shall bleed, ("Love's Sacri-
fice," act iv. scene 1.); an allusion to
the old superstitious practice of per-
forming the operation of bleeding only
on certain days of the moon

N

Nay ; Set a nap on their cheeks,
("The Fancies, Chaste and Noble,"
act v. scene 2.), a freshness, [...]
applying cosmetics
Niceness, scrupulousness [...]
diseases
Nimble [...]
Nokes [...]
No marvel [...] The Witch of Ed-
monton, [...] scene 2.), a proverb
[...]

O

Owes, owner [...]
hath nothing [...]
takes the wealth of [...]
Once; [...]
("Lover's Sacrifice," act v. scene
once for all
Owes; To him that owes it, ("Love's
Sacrifice," act v. scene 1.), owns, po[...]

Ouzle, the blackbird, a generic term

P

Painter [...] Lover's Melancholy,"
act iv. scene 2. "The Fancies Chaste
and Noble," act ii. scene 1.), the city-
shows [...] the Lord Mayor's day
Panel [...] of various colours
Paralos, an [...]
Paris-Garden [...]
Parramant [...]
Party, [...] alluded to in the
[...] of "The Fancies
[...] and Noble," act v. scene 2.;
[...] man of one hundred and
[...] used in a satiric sense for
[...] wench of [...]
Parts [...] spirit, ("The
Fancies Chaste and Noble," act v.
scene 1.), shares [...]
Peaking, sneaking
Passionate, compassionate; and
("The Witch of Edmonton," act ii.
scene 1.), compassion
Pavin, stately, a grave and majestic
dance
Peevish, silly, perverse, foolish
Physical; As well for physic as per-
fuming, ("'Tis Pity she's a Whore,"
act ii. scene 1.) perfumes of beauty
for accomplishments
Pish (a verb active), destroy
Pink, at a ; A shrewd fellow at a pink,
("The Lady's Trial," act iii. scene
1.) at a thrust or stab, at fighting
Pluck a crow, a low expression for
picking a quarrel
Pomp; Would I might see thee in the
pomp once, ("The Fancies Chaste
and Noble," act v. scene 2.) means,
probably, the procession of the city-
companies
Porter's lodge, ("The Fancies Chaste

4. Tuberculous Meningitis, and other states of cerebral congestion or compression.

5. Arterial Sclerosis (used in its widest sense).

6. Sunstroke.

Thus it will be seen that the three pathological conditions in which it is apt to occur are: States of cardiac failure; states of cerebral congestion; and toxic blood conditions

§ 23. Dropsy is a chronic effusion of fluid into the subcutaneous tissue (when it is known as anasarca or œdema) or into a serous cavity (as in hydrothorax, hydropericardium, ascites). The former, Anasarca, is the variety of dropsy we are now concerned with, for it is a very constant feature of some forms of cardiac disease. General anasarca has to be differentiated from myxœdema, in which the swelling is harder, and does not pit on pressure. It is best to apply the pressure over a bone, such as the lower end of tibia on its inner aspect.

Causes.—The causes of localised dropsies are given in Diseases of the Extremities (§ 455). There are *three varieties of general anasarca*, which differ from each other both pathologically in their origin, and clinically in the course they pursue.

1. **Cardiac Dropsy** (1) *starts*, and throughout the case predominates, in the *most dependent parts*, that is to say, in the legs if the patient has been walking about, or in the back if he has been lying in bed. On inquiry, the patient may complain that the ankles swell towards evening around the top of the boot. (2) Other signs and symptoms of cardiac enfeeblement or dilatation are present; and perhaps those of valvular disease as well. (3) In the history of the case dyspnœa will have *preceded* the dropsy. Dropsy does not occur with equal frequency in all forms of cardiac valvular disease. It is common in disease of the mitral valves, but rare in aortic disease, at any rate until quite the end. The dropsy. which complicates pulmonary disease has the same features as cardiac dropsy, because it is the resulting cardiac weakness which produces the dropsy.

2. **Hepatic Dropsy** (1) always begins and predominates *in the abdomen* (ascites), although the legs may swell subsequently by reason of the pressure of the fluid on the veins within the abdominal cavity. (2) There may be also enlargement or other signs of the liver affection which has given rise to the condition; and if these be absent some other cause of obstruction to the portal vein should be sought (Chapter XII). (3) The dyspnœa will have *followed* the abdominal enlargement.

3. **Renal Dropsy** is (1) *general in its distribution* from the beginning, occurring in the legs and eyelids at the same time, though it is probable that the œdema round the eyes on rising in the morning first attracts the attention of the patient or his friends. (2) Examination of the urine reveals the features of renal disease, but it should be remembered that some degree of albuminuria is a common sign in heart failure. The presence of casts is strong evidence of renal origin. (3) The patient presents a characteristic pale or waxy appearance. In some cases of general

C.M.

anasarca associated with albuminuria the question arises whether the dropsy is of renal or cardiac origin.. This may sometimes be answered by finding the liver enlarged, for this is a natural sequence of cardiac valvular disease, though not of renal disease. The frequent association of chronic renal disease and cirrhosis of the liver must not be forgotten.

Prognosis.—The dropsy of cardiac disease is probably due to the stasis within the veins, and its occurrence is therefore an indication and a measure of the amount of obstruction to the circulation on the right side of the heart.

The *treatment* of all forms of dropsy should be directed to the removal of the cause. Even if this be not removable, the dropsy may frequently be alleviated. The limbs should always be rested, raised to the same level as the body, and kept warm. The additional support of a well-adjusted flannel or stocking bandage is a great comfort to the patient, and helps to prevent further effusion. Diuretics and diaphoretics should be employed. These failing, we may employ direct drainage of the sub-cutaneous tissues. The patient should be supported in an upright posi-tion on a chair so that the fluid drains into his legs. The skin should be painted (without previous washing) with Liquor Iodi, and six to eight small punctures made with a sharp-pointed tenotome, or Southey's tro-cars (two in each leg) may be inserted, and the fluid allowed to drain away. The operation is painless, and has surprisingly good and often lasting effects. Its only danger is the risk of sepsis. Antiseptic absorbent dress-ings should be applied. Six or eight punctures are sufficient, the positions of the veins being avoided. Dropsical limbs have a tendency to the development of eczema, erythrema, cellulitis, and epidemic exfoliative dermatitis, so that strict asepsis should be observed in these procedures.

OBSCURE CAUSES OF GENERAL ANASARCA.—If, in a patient who complains of dropsy, no marked evidences of cardiac, renal, or hepatic disease are discoverable, the following causes may be *suspected :*
1. **Anæmia** is not infrequently attended by some swelling of the ankles at the end of the day. This may appear quite early in chlorosis, but is rare in pernicious anæmia. Swelling of the feet and ankles may be present in the last stages of many exhausting diseases, such as phthisis, in septic states, and in cases of insufficient nutrition and old age. Other defective blood conditions, such as leukæmia, may also be accompanied by dropsy. 2. In **Fatty Heart** anasarca is not a prominent symptom, but a slight degree is frequently present. 3. Among the less frequent causes of dropsy in this country are **Beri-Beri** (§ 598) and **Epidemic Dropsy.** Epidemic dropsy was seen in India first in 1877, and is believed to be identical with Beri-Beri and War œdema. **War œdema** was met with amongst the Turkish prisoners of war and in the famine-stricken areas of Europe from 1916 onwards. There is œdema, anæmia, debility and gastro-intestinal disturbance. Albuminuria may be present or absent. The disease is due to a deficiency of the essential food factors. When complicating factors such as dysentery and malaria were present, improvement did not follow a physiologically correct diet. 4. General œdema, without urinary changes, following a gastro-intestinal upset, has occurred in young children. Some have been cured by hypodermic injections of adrenalin ; other cases have been fatal. Milroy first described a **hereditary œdema** in which a solid œdema of the legs existed from birth, unattended by danger to life. Congenital general œdema is usually fatal.

§ 24. **Palpitation** is the sensation of " fluttering in the chest " experienced by an individual who is conscious of the rapid beating of his heart. It arises under two sets of conditions, due to extrinsic and intrinsic causes. The *extrinsic* group is the larger and less serious ; it includes :—

1. In **Anæmia** the palpitation is a frequent and often distressing feature.

2. In **Dyspepsia** palpitation is often present. In such cases it frequently occurs at night, especially after taking a heavy meal. It may, in these circumstances, be accompanied by morbid dreads— *e.g.*, of impending death— by breathlessness, cardiac pain, and by other cardiac symptoms.

3. Certain **Local Conditions**, such as thoracic or abdominal tumour, or dilated stomach, which hamper the heart's action, may produce palpitation, although the heart be healthy.

4. In **Graves' Disease** (exophthalmic goitre) violent palpitation and greatly increased rate of the heart are prominent features. In quite a number of my cases this and the other nervous symptoms of the disorder had existed for many months, or years, before the two diagnostic features thyroid enlargement and exophthalmos— became obvious. Graves' disease should always be suspected in cases of persistent palpitation for which no cause can be made out. Hyperthyroidism can always be diagnosed by means of a B.M.R. test (§ 668).

5. **Incipient tuberculosis** (§ 110).

6 **Nervous** conditions, such as fright, fear, or other emotion, especially after an exhausting illness. It also occurs in hysteria and neurasthenia.

7. The excessive use of certain **Drugs** or **Articles of Diet,** notably tobacco, tea, coffee, and alcohol.

Intrinsic causes include : (1) gross cardiac lesions ; (2) auricular flutter ; (3) auricular fibrillation ; and (4) Paroxysmal Tachycardia.

These conditions and their differential features are dealt with in Section C (§§ 59 *et seq.*).

§ 25. **Pain in the Chest** is not always present, even in grave cardiac disease. A feeling of discomfort or constriction, or a sense of suffocation, is a symptom frequently present when the action of the heart is deranged by functional or structural diseases— oftener perhaps by functional. The importance of pain as a symptom of heart disease lies in the fact that the onset of discomfort or pain, together with breathlessness, after slight exertion, suggests the presence of cardiac trouble even when physical examination reveals little or nothing. If the pain of which the patient complains is due to cardiac disease or embarrassment, tenderness of the skin and muscles in certain definite situations will usually be present. If this tenderness is not present, the pain is possibly not cardiac in origin.

It is important to realise that the internal organs are only capable of recognising one form of pain-stimulus, namely, that due to tension either of the wall of a hollow viscus or of the capsule of a solid organ. They are insensitive to simple tactile stimuli. The heart is innervated by fibres of the sympathetic system which are in communication with the lower cervical and upper eight dorsal segments of the cord. Stimuli

arising in the heart may keep up a constant irritation of the spinal segments concerned. Any mild stimulus reaching that level of the cord by a peripheral nerve is magnified into a pain sensation ; or if there is marked spinal irritability, spontaneous sensations of pain may be aroused in the cutaneous regions corresponding to the peripheral nerve supply. Such pain is known as a " referred pain," because it is not recognised in the brain as originating in the organ which is really responsible for its source, but is referred to the surface of the body. The radiation of the pain of angina to shoulder, neck, and arm is thus readily explained.

The causes of præcordial pain are :

(a) Arising from **Organic affections outside the heart and pericardium.** Intercostal neuralgia, especially that which precedes or follows herpes zoster ; myalgia ; neoplasms ; pleuritic and lung affections (§ 83) ; spinal caries and carcinoma of the vertebræ, and tumours eroding the bones ; the crises of tabes dorsalis ; aneurysm ; painful affections of the spinal cord and inflammation of its membranes.

(b) The pain may be of **Cardiac origin,** and falls under one of two groups : (i.) UNASSOCIATED WITH cardiac or vascular disease such as may arise from *pressure upon the heart* by a distended stomach or by *reflex pain,* referred from the stomach (chiefly) or uterus (occasionally). Tea and tobacco excess are other causes.

(ii.) ASSOCIATED WITH cardiac or vascular disease, such as arises with Angina Pectoris, severe and mild ; coronary disease ; aortic disease ; muscle exhaustion ; high blood-pressure, and Paroxysmal Tachycardia.

Angina Pectoris is a condition manifested by paroxysmal attacks of pain in the chest, varying in severity from mere discomfort to real agony, with a sense of suffocation and other symptoms (see § 47).

In cases of unexplained pain in the chest, and in the absence of cardiac signs, *mediastinal tumour* or *aneurysm of the aorta,* either of the arch or of the descending aorta,[1] should always be suspected, and an X-ray examination made.

Disease of the heart may also be an indirect cause of pain elsewhere than in the præcordium. For instance, with the engorged liver which is a common result of failure of the right auricle and ventricle, the muscles and skin of the abdominal wall are often extremely tender. A simple cause of epigastric tenderness which must never be forgotten in cases of heart and lung disease is muscular strain of the upper rectus muscle and diaphragm from the exertion of constant cough. Pain in the shoulder, arm, neck and jaw is common in cases of disease of the first part of the aorta ; it is usually accompanied by superficial tenderness.

In the *treatment* of præcordial pain an endeavour should be made to ascertain and relieve the cause, but much relief may be obtained temporarily by the application of an opium or belladonna plaster, belladonna liniment, or glycerine of belladonna.

§ 26. Syncope is suspended animation due to anæmia of the brain. It is often preceded by giddiness, nausea, and a feeling of faintness. The

[1] In a case of aneurysm of the descending thoracic aorta which I have recently seen, almost the only symptom or sign, besides breathlessness, during eighteen months —up to the time of sudden death from rupture of the aneurysm into a bronchus-- was continuous pain in the præcordial region. It extended round from the back on the left side, and was thought to be intercostal neuralgia.

face is ashy pale and the pulse and respiration feeble. Its advent is usually sudden, but recovery, after the attack has lasted some minutes, is gradual.

Diagnosis.– Syncope has to be distinguished from *epilepsy minor*, which it resembles in many respects. First, epilepsy minor (*petit mal*) is usually preceded by an aura, though this is evident to the patient only. Secondly, its advent is more sudden than syncope, and the return to consciousness equally sudden and complete, for the patient in *petit mal* can go on with his usual avocations immediately afterwards. Thirdly, syncope rarely occurs without some definite determining cause, although it may be of a trivial nature—such, for example, as a heated room, or the sight of blood. Finally, in epilepsy minor there is often a history of major attacks at some time.

Causes.– The condition may be brought about in three ways : (1) Deficiency of blood, *e.g.* hæmorrhage. (2) Vasomotor instability, in which there is " pooling " of the blood. This is seen in the common form of faint in which the abdominal vessels suddenly lose their " tone," dilate, and absorb the blood which is needed elsewhere. (3) Inability of the heart to drive the blood to the brain, as in failure of the left ventricle in aortic incompetence, or abeyance of its action as in Stokes-Adams' disease.

The **Vasomotor** group is the largest. The " faints " occur chiefly in young, anæmic, and nervous females and boys at puberty ; who, when exposed to grief, bereavement, or any sudden emotion, or too hot rooms full of vitiated air, develop the familiar " fainting attack."

Predisposing causes are :—(1) Anæmia, debility, hunger, or starvation ; (2) diminished resistance in the peripheral and splanchnic arteries, such as occurs with excessive heat, as in hot rooms or Turkish baths ; (3) sudden assumption of the erect posture, as in jumping from bed, may produce syncope in the aged ; (4) sometimes, in addition to the preceding, the splanchnic veins are suddenly dilated when the intra-abdominal pressure is rapidly lowered, as by emptying the bladder, and this leads to anæmia of the brain and syncope.

As a symptom of **Structural Heart Disease** syncope is a much more serious matter. It is a not infrequent symptom in any form of cardiac disease attended by enfeeblement of the heart's action, and is serious as indicating weakness of the *cardiac wall*. It is more often met with in aortic than in mitral valve disease. It may be the first and only symptom of fatty, fibroid, or other degeneration of the heart (§ 52). Syncopal attacks, preceded by giddiness, may arise in old people who are the subjects of arterial thickening and degeneration, this being the cause of what is known as " senile syncope." So important is it to distinguish between the two kinds of fainting attacks that their differential features are given in a table. In both there is a pallor of the surface, and there may be feeble pulse, though the pulse in nervous faints is sometimes unaltered. Both recover best in the recumbent position.

Prognosis.– Syncope in the young is, as we have seen, usually a neurosis, whereas in the aged it is generally an evidence of cardio-vascular

degeneration. In the former, therefore, it is usually as trivial as in the latter it is serious—the gravity depending upon the nature of the lesion. .

Treatment.—Place the patient immediately in a horizontal position with the head low. This may be most readily done on the floor, but if there is little space, instruct the patient to bend forward and lower the head between the knees. Apply ammonia to the nostrils, throw cold water on the face, and, in severe cases, apply a mustard-plaster over the heart. If recovery does not promptly take place, and the pulse be very feeble, a hypodermic injection of 15 or 20 ℳ of ether or brandy may be resorted to. For further treatment, see Collapse. The underlying cause must be carefully sought for and treated when the patient has recovered from the urgent syncopal condition.

§ 27. Cough is a symptom which belongs more especially to diseases of the lungs (§ 81), but it is met with in diseases of the cardio-vascular system under two circumstances. Firstly, the lungs are very often involved secondarily to the heart, and then the patient has the cough usual to pulmonary disorders. Secondly, when the aorta, by its enlargement, presses on the recurrent laryngeal nerve, a peculiar dry, brassy, or,

<div align="center">TABLE I.</div>

Cardiac Syncope, associated with Structural Derangement of the Heart.	**Nervous Faints,** in which only the Nervous Apparatus of the Cardio-Vascular System is deranged.
Usually adults; both sexes equally affected.	Usually females, young or at menopause. Children or young adults.
May come on without any apparent determining cause, or after excessive exertion.	Some determining cause always present (e.g. emotion), acting on the nervous system.
Not accompanied by emotional manifestations.	Often preceded or followed by crying or laughter, and other emotional symptoms.
May be fatal.	Never fatal.
Evidences of cardio-vascular degeneration and its causes. If no signs, suspect fatty heart.	Sometimes evidences of the hysterical diathesis — e.g., hemianæsthesia, ovarian tenderness, globus, or loss of vaso-motor control.

as it is aptly called, " gander " cough is present, which is so characteristic as to be in itself a valuable diagnostic feature of aneurysm. In pericarditis cough may be troublesome.

§ 28. Cyanosis (κυάνεος, blue) is lividity of the surface of the body. It is not one of the most common symptoms in heart disease, but it is one of the most serious and unmistakable evidences of enfeebled or retarded circulation. It is generally most pronounced on the lips, fingers, nose,

ears, and toes, and the skin may vary in colour from faint purple to almost black. When only a slight degree of cyanosis is present, it may be detected by closely examining the roots of the nails. Cyanosis indicates deficient aëration of the blood, which may be due to (1) stagnation of the blood in the capillaries; (2) defective oxygenation of blood which is normally circulating; (3) abnormal compounds of the hæmoglobin (rare).

Generally speaking, the first is the most common cause in cases where the heart is at fault; the second where the lungs are defective; and the third where there is disorder of the digestive tract. It should be remembered, however, that when the heart is involved the lungs become affected later, and that the first and second causes are to be found in association in many cyanotic conditions.

In the first class the causes to be looked for are:

1. Deficient *vis a tergo*, as in failing compensation towards the termination of many cases of heart disease, or of diseases in which the heart is secondarily affected —*e.g.*, cirrhosis of the liver, profound toxæmia.

2. Constriction of arterioles, as in angio-neurotic cyanosis, exposure to cold and other conditions.

3. Obstruction to the flow of blood through a part, as in thrombosis, when the cyanosis is usually localised.

4. Increased concentration of the blood, as after comparative dehydration from profuse diarrhœa or sweating, and in conditions in which the polycythæmia depends on some defect in the circulatory system necessitating an increased number of corpuscles to provide a sufficient supply of oxygen to the tissue—*e.g.*, congenital heart disease. It is also seen in erythræmia (see below).

In the second class there are four subdivisions:

1. Failure of venous blood to reach the lungs, as in some cases of congenital heart disease where the blood is in part "short-circuited" through a patent foramen ovale. Unless a considerable amount of blood is so short-circuited no cyanosis may be apparent, or perhaps only the roots of the nails will show the characteristic bluish tinge.

2. Obstructed flow in the pulmonary circulation. This is rare, but may occur in congenital pulmonary stenosis, and when a mediastinal tumour is pressing on any of the pulmonary vessels.

3. Deficiency of lung surface available. This is met with in emphysema and in a variety of other conditions of lung disease and of pressure on the lung with collapse of the lung. Pneumonia, phthisis, tumour, and sudden or chronic pleural effusions are examples of this class.

4. Deficient entry of oxygen. Under this heading come cases of stenosis and obstruction of the bronchi or trachea, as in syphilis, spasm, impaction of foreign bodies, and pressure of tumours, such as aneurysm and goitre. Here also come cases of cyanosis due to absence of the proper proportion of oxygen in the atmosphere, as on the tops of mountains or in very ill-ventilated rooms.

§ 29. **Erythræmia** (Synonyms: Vaquez' disease, polycythæmia vera, splenomegalic polycythæmia).—This is a disease in which there is an overgrowth of that part of the marrow in which the red cells are formed. There is (i.) increase in the number of the red cells, which may be as many as 13,000,000 per c.mm. There is an increased viscosity of the blood, and later an increase in its volume. (ii.) The patients are easily recognised by the redness of their complexions, which often deepens to cyanosis, especially in cold weather. All the superficial vessels are dilated. (iii.) The spleen is often very large, frequently reaching to the pubis. (iv.) Subjective symptoms

are headache, vertigo, pains in the limbs, and dyspnœa. A variety is described without enlarged spleen, but with high blood-pressure and arterio-sclerosis. The patients usually die in six or eight years from syncope or cerebral hæmorrhage ; many of them in asylums.

The *Treatment* must have reference to the cause ; but in several cardiac conditions cyanosis, if unaccompanied by dropsy, is a distinct indication for venesection. Even when there is considerable anasarca, great relief may sometimes follow the removal of 10–20 ounces of blood. The inhalation of oxygen is a measure which yields great, though temporary, relief. The stream from the oxygen cylinder should be allowed to play directly on the patient's nose and mouth. The gas is given preferably through a mask, for frequent periods of 15 minutes. In erythræmia, bleeding gives temporary relief when the blood-pressure is high ; when the blood-pressure is low its action is not so certain. Inunction of mercury over the spleen is useful, and benzol as used in leukæmia (*q.v.*) gives the best results. Arsenic is contra-indicated.

§ 30. In the third class come two rare diseases : sulph-hæmoglobinæmia and methæmoglobinæmia.

Sulph-hæmoglobinæmia (Synonym : Enterogenous cyanosis).—This disease has hitherto been named according to each observer's idea of its etiology, without discriminating it from the not dissimilar condition, methæmoglobinæmia. The most prominent symptom is (i.) cyanosis of a greyish hue, combined with pallor; (ii.) some form of intestinal disorder, usually constipation, occasionally alternating with offensive diarrhœa ; (iii.) extreme weakness ; the patient may appear to be on the point of death for long periods of time. The etiology is uncertain but the disease seems to be associated either with the presence of conditions (possibly bacterial) which allow of the formation of some easily assimilable compound of sulphur which is not normally present, or else with lesions of the mucosa of such a nature that the normal sulphuretted hydrogen of the intestine is given a chance of combining with the blood. The prognosis is good if the morbid intestinal condition reacts satisfactorily to treatment. Intestinal antiseptics must first be tried, and if these fail operative measures may be adopted. Appendicostomy or even excision of the colon may be required.

Methæmoglobinæmia is a condition in which methæmoglobin is found in the blood. The most prominent symptom is (i.) cyanosis ; the tint is a bright blue and there coexists marked pallor. (ii.) Offensive diarrhœa. (iii.) Weakness, dimness of vision, vague pains, and a feeling of collapse. There are from time to time exacerbations of all symptoms with extreme dyspnœa, and at such times the patient seems to be at the point of death. There may also be periods, varying from a few minutes to hours, during which the cyanosis entirely disappears. (iv.) There is usually an antecedent history of drug-taking, or of working in mines, or in the manufacture of explosives. The drugs or fumes which give rise to this type of cyanosis are aniline derivatives (" antikamnia " and " daisy " headache powders, antipyrin, antifebrin) and benzene compounds. Pot. chlorate and some other drugs have a similar effect, but are less often found to be in operation. Many cases have been traced to the presence of aniline dyes in yellow boot polishes. In another class of case there is no drug history, and bacteria have been isolated. In two recorded cases the organism was of the coli group ; in one it was isolated from the blood, and in the other from a pelvic abscess. In some cases of poisoning when a large dose has been taken, as with suicidal intent, the onset of the illness is very acute.

The *diagnosis* must be based on the history and the peculiar cyanosis, but can only be verified by spectroscopic examination of the blood (Fig. 118). The *prognosis*

is bad in acute cases ; in others it depends on the ease with which the intestinal disorder can be treated, and on the discovery and cessation of any causative drug. As regards *treatment*, the cause must be removed. The usual measures for intestinal antisepsis should be tried, and operative procedures may be required.

A **Sallow Hue** of the skin is common in *aortic* valvular disease, which in this respect presents a marked contrast to the cyanotic flush of patients with advanced *mitral* valvular disease. This sallowness is distinguished from jaundice by the absence of the yellow colour from the eyeballs and the absence of bile in the urine. True jaundice, however, does arise in cardiac disease, as a result of the hepatic congestion which is often met with in mitral but not in uncomplicated aortic disease.

Pyrexia and its concomitant symptoms (see Chapter XV) are present in most of the *acute disorders* of heart and pericardium. The temperature in malignant endocarditis is usually of an intermittent or remittent type, with an irregular range, such as that met with in other forms of septicæmia.

§ 31. Sudden Death, or death, say, within a few hours of the apparent commencement of the illness, is a frequent mode of termination of disease of the heart, and it may be the first symptom of disease of this organ. The chief conditions under which sudden death occurs are as follows. The first six of these have reference to the cardio-vascular system.

1. Among the various forms of *cardiac valvular disease*, sudden death is more frequent in aortic than in mitral disease. But sudden death, and, in general terms, the prognosis, depend more upon the condition of the wall than of the valves.

2. It is liable to occur in all forms of disease of the *cardiac wall* - *e.g.*, fatty and fibroid heart (§ 52).

3. It is a common termination to *aortic aneurysm* (§ 65).

4. A patient may die with the first attack of *Angina Pectoris* (§ 47).

5. Sudden profuse *hæmorrhage*, internal or external.

6. *Embolism* of lung, heart chambers, or coronary vessels- *e.g.*, from air in the veins (as in the performance of transfusion), or clots passing to or formed in the heart.

7. The conditions which cause *Coma* may also result in death, which is relatively sudden (§ 564).

8. Nerve diseases which in their progress involve the *medulla* terminate suddenly ; and thus, among the rarer causes, atlanto-axoid disease and syringomyelia may be mentioned.

9. Sudden emotion, injuries to the head, and other conditions acting on the *nervous system by shock* (§ 561).

10. Suddenly-acting *poisons*, such as prussic acid, a large dose of morphia or carbolic acid, aconite, veratria, etc.

11. Sudden rupture of a large cyst, an internal organ, acute disease of the suprarenals, or other cause of *Collapse* (§ 192).

12. Foreign bodies in the trachea, or other causes suddenly stopping the respiration *(asphyxia)*—*e.g.*, Reflex apnœa from irritation of the pleura.

13. Acute pulmonary œdema (§ 97).
14. Lymphatism.

§ 32. **Lymphatism** (*Status Lymphaticus*) is a rare condition frequently unrecognised during life, but it is important as being a cause of sudden death. There is overgrowth of the thymus gland and of the lymphatic tissues throughout the body. There may be no symptoms, the first evidence of the existence of the condition being death after a trivial shock, such as a plunge into a cold bath, a hypodermic injection, or the first touch of the knife in a minor surgical operation. Occasionally death is preceded for months by attacks of dyspnœa, cyanosis, syncope, and convulsive seizures. The physical signs are often indefinite, consisting only of hypertrophied tonsils and adenoids, and the patient is flabby and pale. In other cases the enlarged thymus causes dulness over the upper part of the sternum, the spleen is palpable, and there may be overgrowth of adenoid tissue at the base of the tongue. Subjects of this diathesis must be guarded against any sudden shocks, or exertion, and must be warned against rapid movements of the head and swallowing large unchewed morsels of food. Operative measures which aim at drawing up the thymus from the thoracic inlet have been successfully performed, but the danger of death under operation has made surgeons reluctant to touch these subjects. X-ray applications are the method of choice and appear to be very successful.

PART B.   PHYSICAL EXAMINATION.

§ 33. **Landmarks of the Chest.**—There is a *ridge* on the sternum formed between the manubrium and the gladiolus; it can always be felt opposite the second costal cartilage; and the other ribs can be counted from the second one. The *nipple* is usually situated just external to the fourth costal cartilage, near its junction with the rib; it should correspond to a vertical line dropped from the middle of the clavicle. At the back, the *lower angle* of the scapula is near the seventh rib; and the *scapular line* is a vertical line drawn through the inferior angle of the scapula. The position and relations of the heart can be studied in Fig. 9, which is a sketch taken from the cadaver. The various regions of the thorax, named for convenience of reference, are given in Fig. 33 in the chapter on Pulmonary Diseases.

Inspection.—The appearance of the "heart" case is often characteristic. The well-developed, overgrown, robust appearance of the *young* aortic, with his pale lips, throbbing neck, and over-development of muscles, etc.; the "red" face of acquired mitral stenosis; the small, undergrown, reddish-blue "congenital mitral stenosis"; the "pinched" patchy face, with the tortuous temporal arteries, and the often wasted body, typical of cardio-vascular degeneration; the yellowish anxious face of infective endocarditis; the large white face of renal disease; the blue face of congenital or failing mitral disease; the worried appearance of the patient with angina, or the distressed pale face of pericarditis, can often be recognised at a glance, or at any rate the underlying condition can be very shrewdly guessed.

The respiratory rate, depth and rhythm, cyanosis, engorgement, pulsation or otherwise of the jugulars, presence or absence of carotid pulsation, thyroid enlargement, clubbing of the fingers and, if present, whether

blue (congenital heart disease) or white (infective endocarditis), swelling of the ankles should be noted.

The abdomen should be examined for distension, and the importance of ascertaining such distension, with its serious cardiac embarrassments, should not be under-estimated. The presence of engorged veins, absence of respiratory movement (? pericarditis) and the presence of ascites should all be observed.

Should the patient be confined to bed, particular attention should be directed to the position in which he lies or which he assumes. When the chest has been exposed, its shape and movements, any bulging of the precordium (cardiac disease in early life before the chest has ceased growing) should be noted, also the apex-beat, position and character, special attention being directed towards whether it is heaving (the true sign of cardiac hypertrophy) or slapping and diffuse in character. Systolic recession, indicative of adherent pericardium, should be looked for, not only in the region round the apex, but in the region of the epigastrium and also in the back (Broadbent's sign).

§ 34. **Palpation and the Localisation of the Apex** (see Figs. 7 and 10).— The apex-beat is the farthest point to the left at which the cardiac impulse is distinctly felt, and after inspection, by which it can frequently be seen, should be first palpated by the flat of the hand, and then localised with the finger tips. In an adult male it is normally situated in the fifth interspace about 1½ inches below and ½ inch to the inner side of the nipple line, at a distance of about 3 inches from the mid-sternal line. *These and other cardiac measurements vary with the age* [1] *and proportions of the patient*—facts which are apt to be forgotten. The *most external* portion of the apex-beat should be marked by a dot with an aniline pencil. The principal features to observe about the apex are—its POSITION, CHARACTER and FREQUENCY. The character of the apex beat varies according to which ventricle forms the clinical apex of the heart. The beat of the left ventricle is felt as a forward thrust; that of the right produces a sinking in of the heart away from the chest wall. This, however, is not constant. It is important to bear in mind that the apex beat is considerably modified if the apex happens (as is not infrequent) to pulsate precisely behind a rib. Only when the apex beats in an intercostal space can these three features be satisfactorily noted : this fallacy should be remembered and allowed for. The apex can sometimes be felt more distinctly when the patient leans forward. In dextrocardia the apex is on the right side.

Roughly speaking, two types of apex beat can be recognised : (1) heaving ; (2) slapping. A *heaving* apex beat can be recognised by the forcible lift which the hand experiences at each systole when

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[1] The position of the heart is considerably modified in childhood. The left border comes out to the nipple line, and the right border extends to the right edge of the sternum ; the apex beats almost directly below the nipple, behind the fifth rib, or may be in the fourth interspace.

pressed over the chest. It is *the* sign of cardiac hypertrophy and is typically met with in cases of aortic regurgitation, renal disease, arteriosclerosis in the young and, with modifications, in adherent pericardium. The *slapping* apex beat means a poorly contracting left ventricle, and this occurs under three sets of conditions : (1) When the ventricle is badly filled, badly stretched and therefore badly stimulated, and consequently contracts badly ; *e.g.*, mitral stenosis. (2) When the muscle has degenerated ; *e.g.*, myocarditis, fatty heart, etc. (3) When the muscle is poisoned ; *e.g.*, toxic myocarditis.

In *hypertrophy* of the left ventricle the apex beat is displaced downwards and outwards, and the cardiac impulse is forcible and heaving. In hypertrophy of the right ventricle there is pulsation in the epigastrium and in the lower interspaces, but the apex is in its normal site. With *dilatation* the impulse is diffuse and wavy. The apex is *displaced downwards* in cases of emphysema or pleurisy with effusion ; if the latter be on the left side, the apex may even be displaced beyond the right border of the sternum (see Fig. 41). The apex is displaced *upwards* in pericardial effusion, retracted lung, abdominal tympanites, or with any abdominal tumour pushing up the diaphragm. The apex beat is *obscured* by very muscular or adipose chest walls or emphysema. It is *feeble* with fatty heart ; *wavy* in pericardial effusion and ventricular dilatation. With pericardial adhesions there is a *systolic retraction* of one or more interspaces ; with hypertrophy of the heart a similar condition may be seen near the apex.

The apex rate should be counted and carefully compared with the pulse. Where the beats are equal, apex and pulse rates coincide, but in certain conditions where the beats are unequal, *e.g.*, certain types of extrasystole or auricular fibrillation, apex and pulse rates are different. The difference between the two indicates the number of ineffective beats tending to exhaust the myocardium.

THRILLS.—The presence or absence of thrills should be noted. If present, they should be timed with the carotid and their exact position noted ; observe also whether they are constant or intermittent.

The commonest thrills are : (1) The apical presystolic thrill of mitral stenosis, which may be either constantly present as it is during the stage of full " compensation," when the auricle is well hypertrophied and its contractility unimpaired ; or intermittent, being possibly present only when the patient is lying down, or after exercise. Usually the presystolic thrill is associated with a regular rhythm, but occasionally it exists with an irregular rhythm, due to either auricular extrasystoles or auricular fibrillation. Roughly speaking, an intermittent presystolic thrill, with a regular rhythm, occurs early in mitral stenosis, before the auricle is fully hypertrophied ; intermittent presystolic thrills with an irregular rhythm are found at the commencement of failure, *i.e.*, when the auricles are either throwing premature beats or fibrillating. (2) A systolic thrill at the apex (mitral regurgitation),

pulmonary base (pulmonary stenosis), aortic base (aortic stenosis or aneurysm) of a pericardial friction·may be present.

Any abnormal pulsation should be noted and investigated. Special attention should be directed towards the liver, and the spleen should also be palpated. As regards the arms the condition of the brachials is of tantamount importance. Note whether they are visible, thickened ; if the latter, note whether the thickening is uniform or otherwise, bearing in mind that if moniliform in character, this usually indicates involvement of the muscle coat as well as of the intima. The locomotor artery always signifies two conditions, viz., a rigid vessel and a hypertrophied heart (J. S. G.).
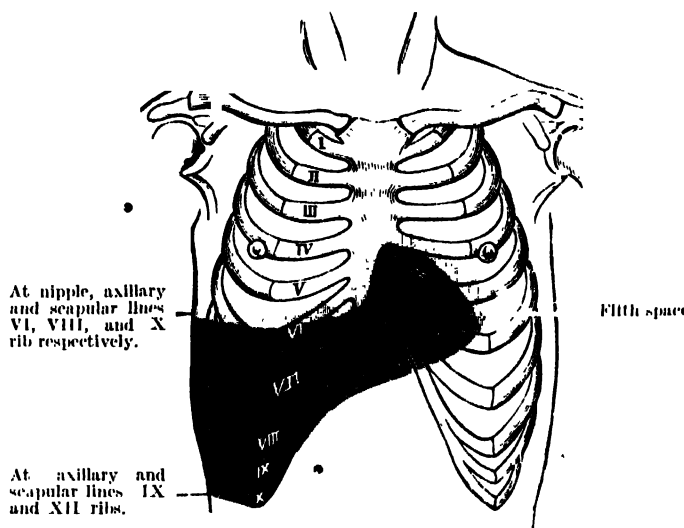
Fig. 7. **Superficial and Deep Dulness of Heart and Liver.**-- The superficial area of cardiac dulness is a triangular one, with the apex upwards. The measurements of this area in a person of average size are 3½ inches transversely, and 2½ to 3 inches vertically along the left border of the sternum. The right border begins at the level of the *fourth costal cartilage*, and corresponds to a vertical line drawn slightly to the left of the middle line of the sternum. The left limit starts from the same point, and runs outwards along the lower border of the fourth costal cartilage to nearly its junction with the rib, then bending downwards to apex-beat. The lower limit is continuous with the liver dulness.

§ 35. **Percussion** of the *superficial* area of the præcordial dulness-- *i.e.* area not covered by lung. In mapping out this area, the percussion stroke should be *very much lighter* than that applied when examining the lungs, liver, spleen, and other deep-seated solid organs (see Fig. 9). The superficial area which is here referred to is a triangular one, with the apex upwards. The measurements of the dull area in a person of average size are 3½ inches transversely, from the mid-sternal line ; and 2½ to 3 inches vertically along the left border of the sternum. Its boundaries are given in Fig. 7. The percussion note over the sternum is very different to that elicited over the chest beside it- it is of a much higher

pitch—consequently, we cannot compare the percussion note in these two situations. We ought, therefore, to percuss upwards and downwards in a vertical line along the sternum to ascertain if any part of it is duller than normal.

The area of *deep-seated* cardiac dulness is ¼ inch larger on each side, and 1 inch larger upwards, than the superficial area. Its limits are considered by most physicians to be less influenced by non-cardiac causes, and therefore more useful for diagnosis, than the superficial area.

Method.—The student should lose no opportunity of PERCUSSING THE NORMAL heart and of attending to the following points : (i.) *Having first localised the apex-beat*, begin outside the cardiac area in a perfectly resonant area. The middle finger of the left hand should be placed flat and *firmly* upon the chest wall parallel to the margin of dulness to be made out, and moved ¼ inch at a time, *always parallel to that margin*, towards the centre of the heart. (ii.) Use only one finger—the second of the right hand—as a hammer, making a short sharp tap with the finger *tip*. The percussing finger should rebound immediately—"staccato," as pianists say. The movement should be made from the *wrist*, or from the knuckle (metacarpo-phalangeal joint), as in playing the piano, and the tap should be a light one. (iii.) By listening attentively to the sound elicited, it will be noticed that it is dull and flat over the heart, like that produced by striking any solid object ; but louder and more resonant outside the area, like the sound produced by striking an empty barrel. It is only possible to define in this way the right, the upper, and the left limits of the dull area, because at the lower limit the cardiac dulness is continuous with that of the liver. Mark with a blue aniline pencil the right or sternal border in two places. The curved upper and left border of the dulness should also be marked by a pencil in two positions —viz., close to the left side of the sternum, and in another place near the nipple ; these can then be joined and continued to the apex-beat.

FALLACIES.— It should be remembered that cardiac enlargement may be *obscured* by the hyper-resonance of emphysematous lungs, and in these circumstances enlargement of the heart or pericardium is very difficult to make out. We have then to rely upon other means than percussion. On the other hand, cardiac enlargement may be *simulated* by a fibrous retraction of the left lung, the heart, nevertheless, remaining of normal size ; or, thirdly, the heart may be *displaced* by an aneurysm or other mediastinal tumour pushing forward, and making the præcordial area appear larger. One or other border of the area of dulness may be *obscured* by pleuritic effusion. Ascites, pleural effusion, or abdominal distension may actually *displace* the heart (see § 97).

§ 36. The Pulse.—At this stage, one may well investigate the arterial Pulse. The radial is the one commonly selected. The usual method of palpating the pulse is to place three fingers of the right hand on it, when the following points can be systematically investigated : *(a) Rate.* Whether abnormally fast or abnormally slow. *(b) Rhythm.* Whether regular or irregular ; if the latter the nature of the irregularity should be investigated. For the sake of convenience we may divide irregular pulses into (1) regularly irregular or (2) irregularly irregular. *(c)* The *Force* (estimated by the impact against the finger) depends upon the condition of the myocardium, *e.g.*, in aortic regurgitation, where the

left ventricle is hypertrophied, the force is considerable, whereas in mitral stenosis, where the left ventricle exercises but little force, the impact is very slight. (d) The Volume, estimated by the lift and duration of the wave, gives one the output of the heart. While (e) the Tension is estimated by the obliteration force and indicates blood pressure.

The most common regular irregularities are : (1) Pulsus bigeminus or pulsus trigeminus (where the pulse goes in twos or threes followed by a pause), indicative of myocardial irritability ; (2) sinus arrhythmia (where the pulse speeds up during inspiration, and slows down during expiration), indicative of (according to some but not the writer) a healthy myocardium ; and (3) pulsus alternans (where big beats and little beats alternate at regular intervals), indicative of failing contractility. This is a sign of very grave prognostic significance.

The commonest irregular irregularities are : (1) The perpetually irregular pulse due to auricular fibrillation. Here the beats not only follow one another at irregular intervals, but are of unequal strength and volume. In addition the pulse-rate differs from the apex-rate, while the irregularity is increased by exercise (§ 61). This condition may be due to mitral stenosis, myocardial degeneration or myocardial toxœmia (Graves' disease). (2) The irregularity due to irregularly occurring extrasystoles (§ 58). This indicates myocardial hyper-irritability, resulting from fatigue, inflammation or degeneration.

§ 37. Auscultation.—For auscultation much practice is required, and the student should never miss an opportunity of listening to the sounds of the heart, *particularly the normal heart.*

The normal heart sounds are three in number—the *First*, or systolic sound, is long, dull and booming in character, and occupies $\frac{4}{10}$ of a second. It is best heard over the region of the apex-beat, *i.e.*, left fifth intercostal space just internal to the nipple line. It is due to two factors : (1) the contraction of the ventricular muscle, (2) the vibrations set up in the auriculo-ventricular valves and chordæ tendinæ by the rising tension in the ventricles. The *Second* or diastolic sound is short, sharp and flapping, occupies about $\frac{2}{10}$ of a second and is heard at the base on a level with the second costal cartilage. It has two components, being produced by the closure of the aortic and pulmonary semilunar valves. The *Third* sound is also diastolic in time, is seldom audible by the ordinary stethoscope, but can be easily detected by means of the cardio-phonograph. Its origin is doubtful. In diseased or damaged conditions of the heart not only are the normal heart sounds modified in various ways, to be described below, but adventitious sounds, murmurs or bruits, are liable to be produced either at the valve orifices or on the surface of the heart (exocardial). In auscultating the heart, therefore, one should pay attention to (a) the characters of the normal heart sounds, and (b) the presence and character of any abnormal sounds (murmurs).

ALTERATIONS OF THE HEART SOUNDS AND THEIR SIGNIFICANCE.—AT

THE APEX. From what has been said about the origin of the First sound, it is clear that its character will be modified by any condition which interferes with the contractility of the muscle or the closure of the auriculo-
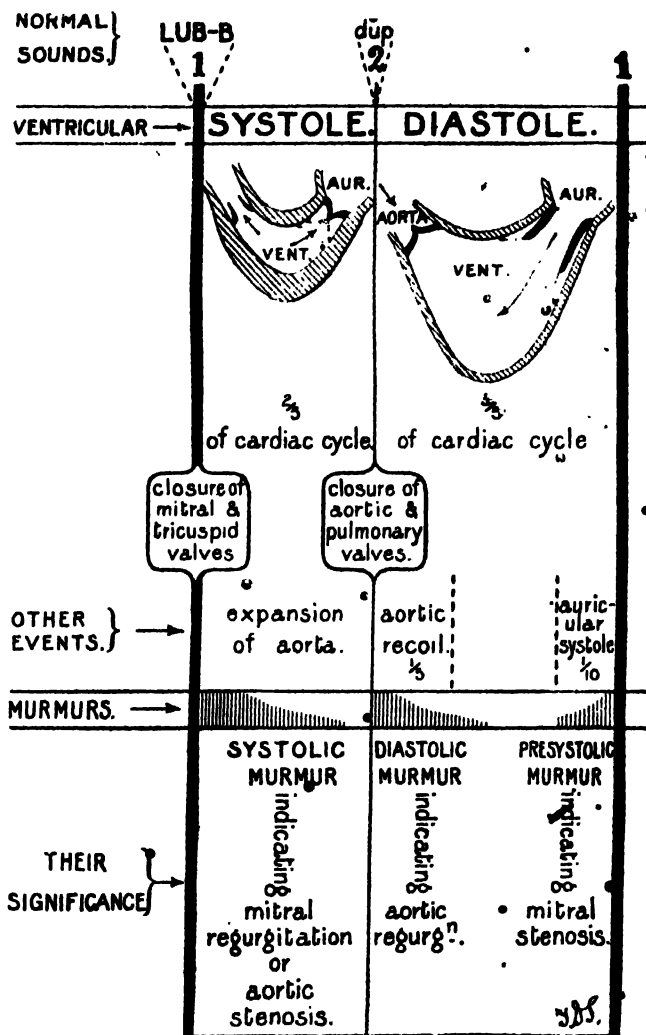
FIG. 8.—Diagram of a Cardiac Cycle, showing various events and their duration, how the different murmurs are produced, and their clinical significance. The student should study this and Fig. 12 very closely.

ventricular valves. This modification may be: (1) shortening, when the First sound becomes somewhat similar to the Second sound, *i.e.*, short, sharp and more valvular in nature. This indicates a poorly contracting ventricle, due to inflammation, degeneration, toxæmia or non-

Internal carotid artery.
Vagus nerve and recurrent laryngeal
Phrenic nerve.   [branch.
Thoracic duct.
Left subclavian artery.
Left subclavian vein.
Ductus arteriosus.
Pulmonary artery.
Pulmonary vein.
Bronchus.
Auricular
[appendix

Above.
L. Innom. vein.
Innom. artery.
L. Carotid a.
L. Subclavian a.

Front.   Behind.
L. Pleura.   Trachea.
L. Vagus.   Trans.   Cardiac plex.
L. Phrenic.   part.   Œsophagus.
Cardiac   Thoracic duct.
nerves.   L. Recurrent n.

Below.
Pulm. art
Duct. arter.
L. Recur. n.
L. Bronchus.

Front.
Pulm. art.
R. Auric. app.
Thymus Gld. and
Pericard.

Right.   Left.
Sup. Vena   Ar-   Pulm. art.
R. Auricle. cend
             part.

Back.
R. Pulm. vessels.
Root of R. lung.

Front.
Pleura.
Root of L. Lung.

Right.   Left
Œsophagus.   Des-   Pleura.
Thoracic D.   cend
              part.

Back.
L. side body of the 10 Dors. Vert.

FIG. 9.—**The Heart and Great Vessels in Situ,** with lungs turned back, sketched from the cadaver. Right ventricle forms greater part of the anterior surface of the heart. Above and to right of this is the right auricle, into which the superior vena cava opens, which collects the blood from the two innominate veins. Passing out from and above the right ventricle is the pulmonary artery, above which again is the remains of the ductus arteriosus, connecting it with the arch of the aorta. Just to the left of the pulmonary artery the left auricular appendix peeps round the corner. The arch of the aorta is seen coming forward from the left ventricle (which is at back, and therefore only seen at left margin of heart), and from its upper convexity arise in order the innominate artery, left carotid, and left subclavian. The trachea is seen behind the vessels, and the phrenic and vagi nerves are seen at the sides, those on the left passing down in front of the aorta behind the root of the left lung. The relations of the ascending, descending, and transverse portion of the aorta are given diagrammatically above.

C.M.   E

stretching, *e.g.*, mitral stenosis. (2) Reduplication, due to a non-synchronising closure of the auriculo-ventricular valves. (3) Weakening or suppression due to pericardial effusion, emphysema, myocardial degeneration, etc., or (4) modification, partial or complete replacement by a murmur or adventitious sound.

The second sound at the apex, due to the closure of the aortic and pulmonary semilunar valves, may be (1) distinct ; (2) reduplicated, when the normal relationship between the pulmonary and systemic tensions are altered, and when the aortic and pulmonary valves do not close simultaneously ; (3) modified by the presence of a murmur as in mitral stenosis, or (4) accentuated when the systemic or pulmonary tension is abnormally high.

AT THE BASE, the *aortic second* sound, normally short, sharp and flapping, due to the closure of the aortic semilunar valves, may be (1) accentuated—indicative of a high peripheral resistance and a high blood pressure ; (2) ringing, indicative of atheroma of the aorta usually with dilatation and rigidity of the valves. This condition, most characteristic to those who are familiar with it, differs from an accentuation and has another significance. It may, or may not be, associated with a high blood pressure. (3) Absence of the aortic second sound means either that the aortic valves do not close owing to injury or destruction, or that they close so quietly that they do not produce an audible sound. This latter condition sometimes occurs in a more or less acute aortitis (J. S. G.). (4) The aortic second sound may be modified by the presence of a murmur which replaces it partially or entirely.

The Pulmonary second sound, due to closure of the pulmonary valves, is also short, sharp and sudden, and in adults less distinct than the aortic second sound. In young children the reverse is the case— the pulmonary second sound being louder than the aortic. It, in turn, may be accentuated (high pulmonary tension), as occurs in mitral stenosis and various lung conditions ; reduplicated, as also occurs in mitral stenosis and other pulmonary defects, or modified by a murmur.

Auscultation should thus provide one with much accurate information regarding the state of the myocardium and the functioning of the different valves. In a healthy heart the intensity of the first sound at the apex bears a definite relationship to the intensity of the aortic second sound at the base. This relationship can be easily ascertained by means of a differential stethoscope. In a healthy heart the ratio is 2--1. Should the myocardium be degenerated, there is a ratio approximately of 1 1. The differential stethoscope is thus an additional means of investigating and assessing the condition of the myocardium.

MURMURS.—Murmurs may be either systolic, presystolic or diastolic in time. The latter, moreover, are frequently divided into early, mid, and late diastolic according to the time at which they occur in the diastole. Further, murmurs may be produced either at the valve orifices (when they are spoken of as endocardial), or outside the heart, of exocardial

origin.   Endocardial murmurs are of two kinds: (*a*) those indicative of structural damage to the valves (organic), and (*b*) those indicative of softening or loss of tone in the auriculo-ventricular rings (atonicity or functional murmurs).   Endocardial murmurs may generally be differ-
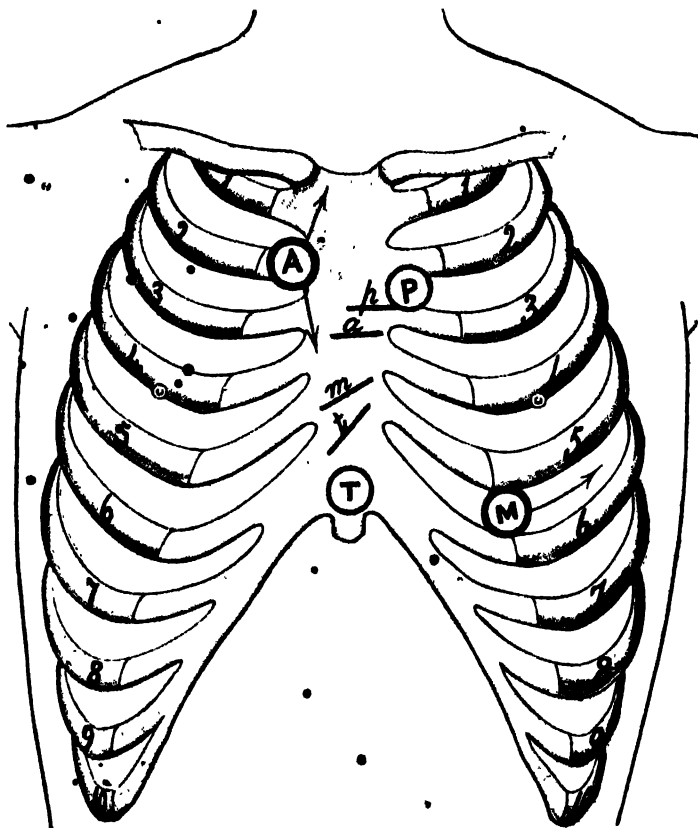
FIG. 10.—DIAGRAM SHOWING THE situation OF THE Cardiac Valves AND THE position IN WHICH THE SEVERAL murmurs ARE HEARD LOUDEST.

p — Pulmonary orifice, at level of upper border of third left costal cartilage.
a — Aortic orifice at level of lower border of third left costal cartilage.
m — Mitral orifice at level of lower border of fourth left costal cartilage.
t — Tricuspid orifice at level of fourth interspace, lying obliquely behind the sternum.
The positions where the sounds produced at the various orifices are best heard are indicated by the letters enclosed in circles.   The arrows mark the direction in which murmurs produced at the corresponding orifices are conducted.
M, Mitral murmurs are best heard at the mitral area—*i.e.*, the apex.
A, Aortic murmurs are best heard at the aortic area—*i.e.*, second right costal cartilage.
P, Pulmonary murmurs are best heard at the pulmonary area--*i.e.*, second left intercostal space.
T, Tricuspid murmurs are best heard at the tricuspid area—*i.e.*, at lower end of sternum.

entiated from exocardial murmurs by the following points: (1) *Endocardial* murmurs are best heard at definite points corresponding to the valve orifices; (2) are conducted or propagated in a definite direction; (3) are harsh or blowing in character.   *Exocardial* murmurs are: (1) superficial and appear to be heard just under the stethoscope, (2) are

usually not heard only over the valve area, (3) are not propagated in the same definite directions, (4) are not necessarily truly systolic or diastolic in time, (5) are often modified by pressure by the stethoscope.

The Atonicity Murmur.—This murmur, which may be heard over either apex or base, is soft and blowing in character and very variable. It may be more or less constantly present, standing, lying and after exercise. If present at the apex, it may be local or conducted outwards to the axilla. It may be absent at rest, and only produced by exercise. On the other hand, it may be present at rest and disappear after exercise. This atonicity murmur, so called, indicates lack of tone in those rings of muscle which support the mitral and other valves, and it is the degree of atonicity present which determines the presence or absence of this murmur under any given conditions.

§ **38. Estimation of Myocardial Efficiency.**—The measure of a heart's efficiency is its capacity for work; this is true of all hearts, whether healthy or diseased. Furthermore, it must be clearly borne in mind that many hearts work perfectly, exhibiting no defects at all, when the patients are at rest, but show serious derangements and definite evidence of myocardial impairment when called upon to do extra work under load. Such defects are usually revealed by the development of irregularities, murmurs, pulsations, etc., which did not previously exist. A simple method of estimating myocardial efficiency is as follows: the patient is weighed and his weight recorded in pounds; he is next made to lie down on a couch; his pulse rate, systolic blood pressure (brachial) and respirations are noted. He is then made to do a definite amount of work in a given time. For ordinary purposes, a convenient test is to make the patient raise his body-weight twenty feet in under thirty seconds; this is easily done by making him run up a flight of stairs of the required number. His pulse, blood pressure and respirations are recorded immediately after his return to the couch. These observations should be repeated every minute until the pulse, respirations and blood pressure are normal. The work done in foot pounds is easily obtained by multiplying the patient's weight in pounds by the height in feet that he has raised himself. The pulse, blood pressure and respirations should, if the myocardium is sound, all increase and be normal again within three minutes. If the myocardium is diseased, the blood pressure will either fail to rise or will actually fall. This principle can be applied for remedial purposes or for ascertaining the reserve energy of the heart (J. S. G.).

EFFECTS OF EXERCISE UPON THE HEART.—(*a*) *The Rate.*—The normal heart responds to exercise by a gradual increase in rate. The increase is more or less uniform, the heart climbing up as exercise is increased. The normal heart rarely speeds up to over 150. It rapidly returns to normal on ceasing the exercise. A soft atonic or poisoned heart responds to exercise by undue acceleration, and very slowly settles down to its normal rate; while in certain diseased conditions of the heart one gets impaired acceleration; the rate practically speaking showing no alteration at all. This may occur in very fast hearts (*e.g.*, auricular flutter), or in very slow hearts (*e.g.*, heart-block). Lastly, in a well-trained physiological heart, such as one meets with in young, highly-trained athletes, the rate does not climb on exercise, but sud-

denly doubles (*e.g.*, at the commencement of the exercise, the rate may be 42, and on exercise suddenly becomes 84)—the so-called athletes' reaction.

(*b*) *Rhythm.*—On exercise the rhythm of the heart may be profoundly modified. (1) An irregularity may be produced, and here I would emphasise the fact that any heart that becomes irregular on exercise is a damaged heart. The most common irregularities brought out by exercise are extrasystoles (indicative of myocardial fatigue), auricular fibrillation (indicative of inco-ordinate auricular contractions secondary to degeneration, toxæmia, etc.), alternation (indicative of failing contractility), a sign of very grave significance. (2) An existing irregularity may be abolished. Practically speaking, the only irregularity which is abolished by exercise is the irregularity due to extrasystoles, *when they are not due to fatigue.* (3) An existing irregularity may be increased. This is true of auricular fibrillation or fatigue extrasystoles.

(*c*) *Sounds.*—Under the influence of exercise, normal heart sounds may be reduplicated or modified by the production of adventitious sounds or murmurs. In early mitral stenosis, and in soft hearts, atonic murmurs may either appear or disappear according to the underlying condition of the myocardium.

(*d*) *Thrills.*—Thrills may be actually produced. This occurs in early mitral stenosis, when increased filling of the auricle results in increased stretching, and
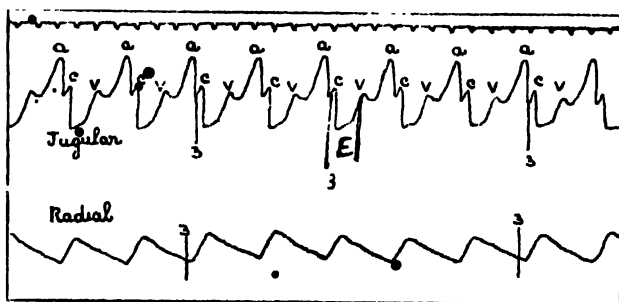
Fig. 11. Simultaneous tracings from the jugular and radial pulses from a patient with a normal heart. The jugular pulse is of the auricular form, and shows a large wave (*a*) due to the auricle. The rhythm is regular. The space E represents the period of ventricular systole. (*Sir James Mackenzie.*)

increased contraction, and so produces a thrill, presystolic of course in time. An existing thrill may be increased under similar conditions or abolished by exhaustion. (J. S. G.)

§ 39. Special Methods of Investigation.- 1. *The Polygraph* is an elaboration of the older sphygmograph. By its means we can take simultaneous, continuous graphic records of two pulsations. In practice it is usual to employ for this purpose the pulsations of the radial artery and the jugular vein, but records of the heart-beat itself (cardiograms) or of the pulsations in an engorged liver, may also be obtained. The importance of the jugular pulsation rests upon the fact that the " bulb " at the lower end of the jugular vein lying between the two heads of the sterno mastoid is not separated by valves from the right auricle ; the blood in the " bulb " and in the auricle is in direct continuity, and changes of pressure in the auricle are recorded as pulsations of the vein. In this way we are afforded a graphic record of the action of the auricle, and can compare it with the action of the ventricle, as recorded by the radial pulse. In health the jugular tracing shows three waves for each wave in the radial pulse. These are known as the " a," the " c," and the " v " waves. The " a " wave is produced by the systole of the auricle. The " c " wave is synchronous with the ventricular beat. It is due to a rise of pressure produced in the auricle in all probability by the bulging of the auriculo-ventricular valves. It was at one time thought to be due to a transmitted wave from the carotid, but this cannot be correct, as it can be obtained after the carotid artery has been dissected away. It occurs in health at a remarkably constant interval of ⅒ of a second after the commencement of the " a " wave. The A C interval is usually taken as representing the conductivity

of the Bundle of His. If the A C interval exceeds ¼ of a second, the conductivity is regarded as being impaired. The " v " wave is often divided and probably is partly systolic and partly diastolic in time. It is said to be due in part to the damming back of the blood in the arteries and veins during the period of the closure of the auriculo-ventricular valves. In pathological conditions of the myocardium, either primary or secondary to valve lesions, the polygraphic record is considerably modified. For example, in the early stages of mitral stenosis where the left auricle contracts forcibly, the " a " wave is abnormally high. In the slightly later stages of mitral stenosis where the left auricle is hypertrophied and somewhat divorced from the right, the " a " wave is drawn out and divided. In partial heart-block the A C interval is increased, while in complete heart-block the " a " wave is dissociated from the " c " and " v " waves. In auricular flutter many " a " waves are said to exist ; in auricular fibrillation the " a " wave disappears.

2. *The Electrocardiograph.*—Electrical phenomena are characteristic of functional or physiological activity, and this is particularly true of such functionally active tissues as gland and muscle. The heart being a very active muscular organ gives out a very definite and characteristic electrical discharge. The electrical phenomena of the heart are remarkably constant in health, but are often profoundly modified in altered conditions of the myocardium resulting either from primary damage or changes secondary to valve lesions. A photographic record of these electrical phenomena is spoken of as an electrocardiogram, and the instrument employed for obtaining it is known as an electrocardiograph. The patient's two arms are supposed to be in contact with the two sides of the base of the heart, while the left foot is in direct connection with the apex ; these are put into electrical connection with the machine. Fig. 12 represents the mean of the curves of the hearts of some healthy students of the Middlesex Hospital School, and may be regarded as the typical curve of a healthy or physiological heart of a young man up to thirty years of age.

Fig. 12.—P — the auricular complex. Q.R.S.T. — the ventricular complexes.

The " P " wave may be regarded as a picture of what the auricle is doing, e.g., when the stimulus starts from the sino-auricular node and is distributed normally throughout the auricle it is represented by a vertical wave exactly as shown in the diagram. Should the auricles be contracting more violently than usual, as in Graves' Disease or early Mitral Stenosis, the altitude of the " p " wave is increased. If the auricles are embarrassed so that they cannot empty freely, and their contraction is prolonged, the " P " wave is opened out and wider than normal (e.g., Mitral Stenosis), while if the two auricles do not quite synchronise in their contraction, the wave is notched (e.g., in Mitral Stenosis or Arterio-Sclerosis). If, on the other hand, the stimulus starts in the region of the auriculo-ventricular node and travels upwards, the wave is inverted. Again, if the auricular muscle does not contract as a whole in a co-ordinate manner, but in more or less inco-ordinated bands (as in Auricular Fibrillation), then the " P " wave is represented by a series of small waves, the size and character of which vary with the nature and degree of the inco-ordination.

Applying the same principles to the ventricular complexes. In lead ii, " R² " generally represents activity in the right ventricle ; hence we find it exaggerated in cases (late) of mitral stenosis or congenital pulmonary stenosis, where the right ventricle is called upon to do extra work.

" S² " is variable, but is usually regarded as being due to activity in the region of the apex, while " T² " represents the terminal phase of ventricular activity, and in all probability corresponds chiefly to activity in the region of the left ventricular (aortic)

base. It is the most prominent feature of the electrocardiogram of young healthy hearts, decreases in prominence in old age, is reduced in amplitude in cases of definite myocardial toxæmia, in cases where the ventricle is not fully distended with blood (e.g., Mitral Stenosis), or degeneration of the ventricular myocardium; while it is delayed or divorced from the " S " or " R " waves wherever the conductivity of the ventricular muscle is impaired from any cause, e.g., cardiosclerosis. The electrocardiographic curve reveals at a glance the time relations of the cardiac cycle, the actual conduction times throughout the different parts of the heart, and also the relative preponderance of the right or left sides, etc. The various irregularities can easily be analysed by this instrument.

3. No examination of the cardio-vascular system can be regarded as complete unless it includes an X-Ray investigation. The technical details must be obtained from a larger textbook ; the main points to which attention should be directed may be thus summarised :—

A. The Heart :—

(1) *Its position* in the mediastinum—its relation to the nipple and midsternal lines and diaphragm.

(2) *Outline and form :*
This is often characteristic, e.g., in young healthy hearts it is vertical, in congenital heart disease round, in old valvular disease globular, but varies with lesion, in senile hearts it is boot-shaped with the long axis horizontal, while in tubercle it is elongated and tubular.

(3) *Size :*
In young normal hearts transverse character approx. 5½, in senile hearts 5¼ to 5¾, in chronic carditis often up to 8 or 9".

(4) *Movements :*
  (a) *The Beat :*
        Regular { ? Tachycardia
                 { ? Physiological.
                 ? Bradycardia { ? Heart Block
        ? Irregular { ? Extrasystoles.
                    { ? Auricular Fibrillation.
  (b) *Effect of Respiration :*
        The concertina heart.
  (c) *Abnormal movements :*
        ? Abnormal or excessive pulsation.
        ? Adhesions give rise to abnormal movements.
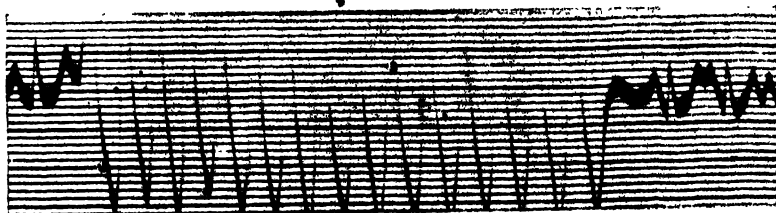
I. Paroxysmal tachycardia due to right ventricular extrasystoles.
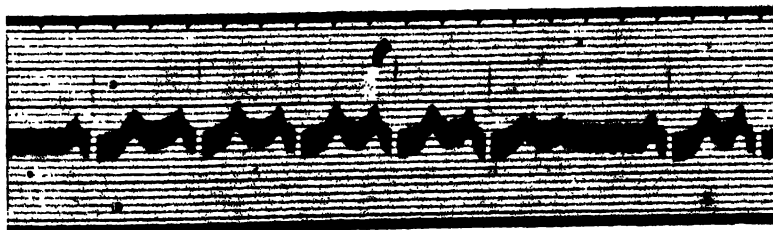
II. Sino-auricular heart block.

FIG. 13.—SOME TYPICAL ELECTROCARDIOGRAMS.

FIG. 13 (*continued*).—SOME TYPICAL ELECTROCARDIOGRAMS.
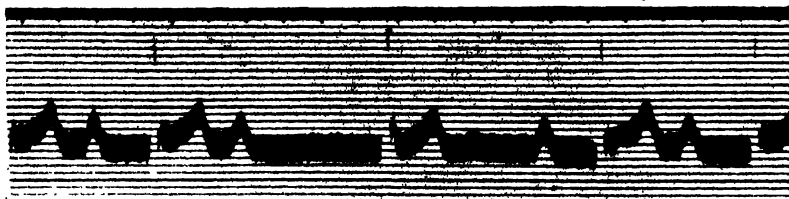
III.   Partial heart block.
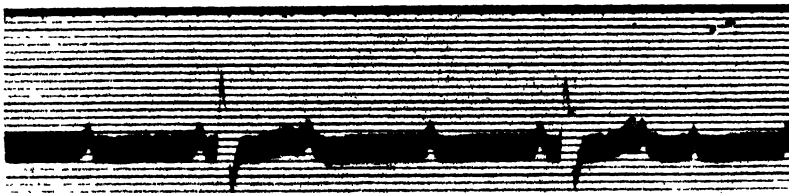
IV.   Complete heart block.
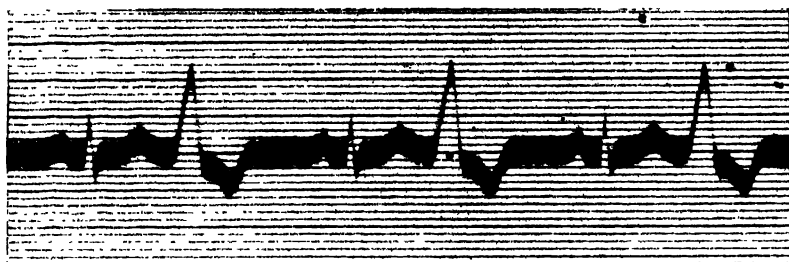
V.   Coupled beats due to right ventricular extrasystoles (digitalis).

VI.   Triple rhythm (right ventricular extrasystoles).
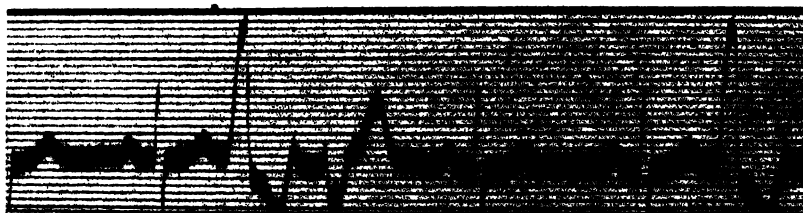
VII.   Right and left ventricular extrasystoles (tobacco).

B. THE GREAT VESSELS :—
    Examine specially in oblique position ; note cardio-phrenic space ; ? dilatation or aneurysm ; ? mediastinitis or growth or foreign body.

C. THE PERICARDIUM :—
    ? adhesions ; ? fluid.

4. *The Orthodiagraph* is an instrument whereby outline diagrams can be made of the actual size of the heart itself, so that accurate measurements can be obtained not only of the heart but also of the great vessels, etc.

PART C. DISEASES OF THE HEART AND PERICARDIUM :
THEIR DIAGNOSIS, PROGNOSIS, AND TREATMENT

§ 40. Classification.— For practical purposes diseases of the heart and pericardium may be classified under five prominent differential features : Disorders WITH PYREXIA ; Disorders in which PAIN is a characteristic symptom ; Disorders which are attended by an ENLARGEMENT of the AREA of CARDIAC DULNESS ; Disorders in which AN ALTERATION of the CARDIAC SOUNDS, or a MURMUR forms the diagnostic feature ; and Cardiac conditions which are recognised by an ALTERATION of the RATE or RHYTHM of the PULSE.

A. PYREXIA . . . . . $\begin{cases} \text{I. Pericarditis.} \\ \text{II. Acute Endocarditis.} \end{cases}$

B. PAIN . $\begin{cases} \text{I. Angina Pectoris.} \\ \text{II. Exhaustion of the heart muscle.} \\ \text{III. Pericarditis.} \end{cases}$

C. ENLARGEMENT OF THE AREA OF CARDIAC DULNESS . $\begin{cases} \text{I. Cardiac Hypertrophy.} \\ \text{II. Cardiac Dilatation.} \\ \text{III. Pericardial Effusion.} \\ \text{IV. Adherent Pericardium.} \end{cases}$

D. ALTERED HEART SOUNDS AND MURMURS $\left.\begin{array}{l} \text{I. Myocardial Degeneration.} \\ \text{II. Endocarditis.} \\ \text{III. Congenital Heart Disease.} \\ \text{IV. Pericarditis.} \end{array}\right.$

E. ALTERATION OF RHYTHM OR RATE OF PULSE .
    I. Sinus Arrhythmia.
    II. Premature Beats (extrasystole).
    III. Tachycardia.
    IV. Auricular Flutter.
    V. Auricular Fibrillation.
    VI. Bradycardia.
    VII. Heart-Block   (Stokes-Adams' Syndrome).

§ 41. The **Routine procedure** in the investigation of a cardio-vascular problem may be considered under the following headings—(1) The origin of the present symptoms, *e.g.*, whether they supervened on any definite illness, acute or chronic, or followed on some definite action, emotion, etc.

(2) The personal history, especially as regards (*a*) previous diseases

such as rheumatic fever, quinsy, or chorea, tonsillitis, growing pains, scarlet fever, influenza, syphilis, etc.; (*b*) habits of life, especially as regards exercise, alcohol and tobacco.

(3) Family history. Certain diseases, *e.g.*, rheumatic fever, arteriosclerosis, etc., tend to run in families, and are known to predispose to heart disease.

(4) Symptoms. The commonest symptoms associated with heart disease are dyspnœa, palpitation, pain, vertigo, faintness, and a sense of exhaustion. These are dealt with in Part A.

(5) Physical examination of the patient (Part B). Inasmuch as many hearts, when only slightly damaged, function normally when the patient is at rest and their "load" is light, but develop obvious defects of action under "load," it is essential to examine the patient three times—standing, lying, and after exercise. Further, it is convenient to divide the examination into: (*a*) Ordinary routine clinical examination, under which heading one would include the results of inspection (§ 33), palpation (§ 34), percussion (§ 35), the pulse (§ 36) and auscultation (§ 37). (*b*) Cardiac efficiency tests (§ 38) used for ascertaining the reserve energy of the heart. (*c*) Special instrumental methods of examination (§ 39).

A. If the symptoms of which the patient complains are unattended by Pyrexia, turn to § 42. If the disease is **attended by Pyrexia**, it is probably ACUTE PERICARDITIS or ACUTE ENDOCARDITIS, either rheumatic or infective in origin.

I. THE TEMPERATURE IS ELEVATED, *the patient is in evident distress, and the præcordial area of* DULNESS IS INCREASED, *the shape of the dulness being* PYRAMIDAL, *with the point upwards. The distress is probably* ACUTE PERICARDITIS.

§ 42. **Acute Pericarditis** is an acute inflammation of the pericardial sac. The disease has two stages: the *first* precedes, and the *second* follows, the effusion of fluid into the pericardial sac. It is not infrequently met with as a primary affection. It supervenes during the course of many different diseases, and the symptoms of these may mask its onset. Rheumatic fever is certainly its most common cause, and it should be remembered that it may be the first manifestation of this affection. We should always examine the heart daily in rheumatic fever, and in acute renal affections, because in these acute pericarditis may come on insidiously, without pain or tenderness, its advent being marked perhaps only by the occurrence of delirium, so rare otherwise in acute rheumatism, or pallor and vomiting, especially in children.

Symptoms.—(1) The patient wears an anxious, troubled look, and the cheeks are flushed or pallid; there are fever and a rapid pulse; the breathing is rapid, and he complains of severe pain over the heart (occasionally referred to the abdomen), increased by pressure, movement, or respiration. Abdominal rigidity may occur. (2) *Physical Signs.*—The

præcordial dulness is only slightly increased at first, but a scratching, harsh *double* friction sound, " to and fro," like a saw, is heard on auscultation. This may be distinguished from a murmur produced *within* the heart by (i.) always being double, *i.e.*, accompanying the movements of the heart, and rarely exactly synchronous with the first and second sounds ; (ii.) the second part of the rub is usually continuous with the first, without any diastolic pause ; (iii.) it is often loudest at the root of the great vessels, over the third left costal cartilage ; (iv.) it varies in its character from time to time, and is increased by gentle pressure with the stethoscope ; (v.) pressure will also elicit another character—viz., that the disease is usually accompanied by tenderness, as well as pain. The differentiation between peri- and endocardial murmurs is so important that it is also given in a tabular form below (Table II). To distinguish pericardial from pleuritic friction is very easy, because the latter ceases if the patient holds his breath. Note that as the effusion occurs the murmur may become less distinct, but it very rarely disappears entirely. It is again intensified as the effusion clears up.

(3) *Second Stage,* or stage of pericardial effusion. The inflammation may subside, but more frequently, in the course of a day or two, effusion of fluid occurs, and the pain and tenderness diminish. The rub becomes less audible, though it can still be heard at the base of the heart. The temperature may fall a little, but the breathlessness and other symptoms continue. A troublesome cough is frequently added, and dysphagia and vomiting sometimes occur. Pulsus paradoxus may be present. *The increased area of dulness,* due to pericardial effusion, may be greater than the enlargement from any other cause. (i.) It is of *triangular shape,* with apex upwards, reaching to the third, or even second, costal cartilage. (ii.) There is often actual or apparent *raising of the position of the apex-beat.* (iii.) The *dulness extends to the left* of the apex-beat. There is progressive weakening of the heart sounds at this time, because they are transmitted through fluid. It is possible by the height of the dulness along the sternum, which should be watched each day, to detect alteration in the amount of fluid present. Ewart describes a square patch of dulness with absence of breath-sounds at the base of the left lung.

Etiology.—Pericarditis may attack any age and either sex, but is almost always preceded and caused by some other disease. It is doubtful if it is ever idiopathic. The causes may be ranged under five heads : (1) *Injury.* (2) *Certain acute infections :* acute rheumatism, pyæmia (staphylococcal, streptococcal, pneumococcal), scarlet fever, variola, typhus, typhoid, and influenza, and some constitutional diseases—Bright's disease, scurvy, gout. (3) *Chronic infections—e.g.*, tubercle. (4) *Morbid growths—e.g.*, cancer. In the two last the process tends to be subacute, and is accompanied by a large amount of fluid, often blood-stained. (5) *Extension* from adjacent disease of organs, among which may be mentioned pleurisy or pleuro-pneumonia, especially on the left side ; intrathoracic aneurysm (pericarditis may be the precursor of rupture into

the pericardium); solid intrathoracic tumours; perforating ulcer of the œsophagus; various diseases below the diaphragm—e.g., abscess. or hydatid of the liver.

Course and Prognosis.—The duration of acute pericarditis with effusion varies widely, but it averages about fifteen to twenty-five days. It may undergo resolution with or without the formation of adhesions (Adherent Pericardium, § 51); or result in chronic effusion (Hydropericardium, § 51); or become purulent (Pyopericardium, § 43 below). Pericarditis with effusion is always a serious malady, but the prognosis depends much on the underlying cause, the amount of distension of the pericardial sac, and the evidences of accompanying myocar-. dial disease—dyspnœa and cyanosis with feebleness, rapidity, and irregularity of the pulse. Pericarditis complicating rheumatism, like the other complications of that disease, tends to recover, but it may leave a weakened heart, and lead to cardiac dilatation. In renal disease it is a serious though often latent affection; and in pyæmia, when it is generally purulent, it adds to the gravity of that serious disorder. In infancy and in debilitated patients it is also grave.
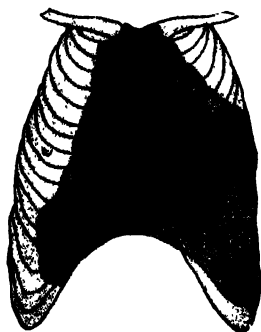
FIG. 14.—DIAGRAM FROM A CASE OF RHEUMATIC PERICARD-ITIS WITH EFFUSION.—Eliza P., aged twenty-seven. Heavy shading corresponds to position of maximum in-tensity of friction. Medium shading corresponds to the area of deep cardiac dulness. Light shading corresponds to the area over which the pericardial friction is aud-ible. It is often taught that one of the features distin-guishing peri- from endo-cardial murmurs is the limitation of the former to the præcordial region; but I have many times satisfied myself that this is not so, and this case is one of sev-eral examples I have met with verified by autopsy.

Diagnosis.—The diagnosis from acute endo-carditis has been considered above, and in Table II, p. 62. It is distinguished from dila-tation by the following points: the left bor-der of the dulness in pericardial effusion extends beyond the apex-beat, and the apex-beat may be displaced upwards; the right border of dulness has a convex outline and the cardio-hepatic angle at the right fifth inter-costal space is dull (Rotch's sign); lack of movement of the epigastrium with respiration is another valuable sign. It should not be forgotten that both conditions may be present at the same time. X-Ray examination, when possible, will usually enable one to make quite sure of the diagnosis.

Treatment.—In the inflammatory stage the patient should be kept in bed absolutely *without movement*, on light fluid diet; and cotton-wool, a poultice, or warm fomentation applied to the præcordium. This usually gives more relief than the ice-bag which was recommended by Dr. Lees, though this undoubtedly relieves the symptoms, controls the restlessness of a young patient, and probably also reduces the heart rate. If the distress is great, wonderful relief is obtained from the application of four or five leeches over the præcordium. If cyanosis, orthopnœa, and rapid small pulse are present, indicating considerable

cardiac embarrassment, bleeding (4 to 6 ounces) is a prompt and efficacious measure. Opium (gr. ¼ quartis horis), or morphia hypodermically, is of great value for the pain and distress. Effervescing salines should be administered. Digitalis mx. is given in frequent doses for cardiac failure, and stimulants, such as brandy and ether, according to the state of the pulse. The effect of digitalis must be very carefully watched, lest it increase the cardiac embarrassment. For hyperpyrexia and delirium some recommend the graduated bath, but the necessary movement is a grave objection. Tepid or cool sponging is a useful means of lowering the temperature, and will often induce sleep.

Treatment for the cause of the pericarditis should be combined with the foregoing—e.g., sodium salicylate combined with alkalies for acute rheumatism; diuretics and hot-air baths for renal disease; quinine in large doses or appropriate vaccines for pyæmia. In the stage of effusion free blistering promotes absorption, but it must be remembered that renal disease is a contra-indication to blistering. If the effusion becomes chronic, potassium iodide (gr. v. (0·3 gm.) t.i.d.) and diuretics may be given (F. 55). Iodine paint and other local counter-irritants are also useful.

PARACENTESIS PERICARDII.—If, at any time, the effusion be considerable, and the cardiac embarrassment very great, as evidenced by severe dyspnœa, and a rapid, irregular, low tension pulse; if leeches and bleeding have failed to give relief, exploration with a hypodermic syringe, under strictly aseptic precautions, may be practised to ascertain the nature of the fluid. If clear fluid be found, paracentesis should be performed, and the operation may be done without fear, if rigid aseptic precautions be employed, and the point of the trocar pushed slowly inwards, so as to avoid stabbing the heart or one of its thin-walled coronary vessels. Incise the integument in the fifth left interspace, close to the sixth rib, 2 to 2¼ inches to the left of the middle of the sternum (in an adult of average size). Insert the trocar and cannula vertically to the surface; withdraw the former directly it pierces the wall. Eight or twelve, or even forty ounces (in a chronic case) may be thus gradually removed. This operation is never necessary in the rheumatic pericarditis of childhood.

§ 43. Pyopericarditis.—Sometimes in debilitated children and in the course of scarlatina, in phthisis and empyema, always in the pericarditis of pyæmia, and under some other conditions, the fluid in the pericardium takes on a purulent or sero-purulent character. This condition is sometimes revealed (as in a collection of pus in other parts of the body) by the occurrence of (1) shivering attacks, (2) profuse perspirations, and (3) a temperature with wide variations in the course of a few hours, in addition to the clinical features of acute pericarditis above described. But it is very difficult to diagnose, because the friction sound is usually absent. It is usually fatal.

Pyopericarditis is the form which pericarditis frequently assumes in infancy, and is then extremely difficult to diagnose. In addition to the small measurements with which we have to deal, the left lung may become adherent to the chest wall early in the disease, and so prevent the recognition of the enlargement of the præcordial dulness. It is only from the profound disturbance of the circulation and the progressive weakness with anæmia and leucocytosis, that we can assume the presence of pus.

Treatment.—Quinine in large doses, cryogenin and like remedies, may be administered, but a large hypodermic syringe, rendered thoroughly antiseptic, should be very carefully introduced whenever the existence of pyopericardium is suspected. If the fluid withdrawn be of a purulent nature, paracentesis, or, better still, free drainage, should be effected.

Pneumocardium is a rare condition in which air reaches the pericardial sac from the lungs or stomach.

Hæmopericardium is very rare. Aneurysm of the first part of the aorta or of the cardiac wall, rupture or wounds of the heart, scurvy and other blood diseases, may lead to sudden death owing to the sudden influx of blood into the pericardium. A small amount of bleeding may be seen in the pericarditis due to Bright's disease, malignant growths, and tubercle.

§ **44. Latent Pericarditis**—*i.e.*, pericarditis without *symptoms* (though not necessarily without physical signs). In most patients in whom we find a pericardial effusion a history of acute pericarditis is obtainable ; but it is a fact not sufficiently recognised that pericarditis may come on quite insidiously, without any acute symptoms. The effusion may be discovered when examining the heart as a matter of routine, or perhaps not until the autopsy. Moreover, I have, in the post-mortem room, on more than one occasion found a totally adherent pericardium in a patient in whom the most careful inquiry had failed to reveal any symptoms pointing to the heart during life. It is a latent pericarditis of this kind which ordinarily complicates RENAL DISEASE. In ACUTE RHEUMATISM also *its advent may be indicated only by delirium or vomiting :* and GOUTY persons also may be attacked by this latent disease after exposure to chill.

Pericarditis frequently results in Adherent Pericardium (§ 51).

We now pass to the other acute disorder with pyrexia.—II. ACUTE ENDOCARDITIS.

II. THE TEMPERATURE IS ELEVATED. *The præcordial area of dulness is not necessarily increased, and on auscultating the chest there is a* MURMUR *added to the heart sounds—the disease is probably* ACUTE ENDOCARDITIS. *It is not always easy to distinguish an endocardial from a pericardial murmur* (see table below).

TABLE II.—DIAGNOSIS OF ENDOCARDIAL FROM PERICARDIAL MURMURS.

Endocardial Murmurs.	Pericardial Murmurs.
1. May accompany first or second sound only, or both. If double, there is always a short interval of silence between the two bruits.	Usually double—always superficial and can be heard throughout the diastole, as well as the systole, without any interval between the two bruits.
2. Loudest in one of the valvular areas.	Usually loudest over third left costal cartilage (root of big vessels).
3. May be conducted into the axilla, or along the aorta and carotids.	Mostly confined to the præcordium.[1]
4. Usually no pain or tenderness.	Often accompanied by pain.

§ **45. Acute Endocarditis** is acute inflammation of the valves or mural endocardium of the heart. It is usually attended by enlargement of the præcordial dulness, because a degree of myocarditis and dilatation is associated with it. In a very large proportion of cases it complicates

[1] For an exception to this, see Fig. 14, p. 60.

some other disease; and, like pericarditis, it is very frequently associated with acute rheumatism; it may even be the first evidence of that disease.

There are two varieties of endocarditis, commonly known as SIMPLE and MALIGNANT, and there are three groups of symptoms found with each.

In SIMPLE or BENIGN ENDOCARDITIS, as in the other variety, (1) the characteristic feature is the *development of a murmur*, usually heard loudest at the apex because the mitral valve is the one most frequently involved in acute rheumatism; but it may be heard in any situation, depending on the valve affected (see p. 51), and it may be single or double in rhythm. The murmur has to be diagnosed from that of pericarditis (see table above), and, if possible, from that due to old valvular disease or to weakening and dilatation of the ring. In the acute disease the murmur is usually softer and heard over a more limited area; in old valvular disease it is harsher, and is conducted in different directions (*vide* Cardiac Valvular Disease, § 53). The previous history, and the presence of cardiac dilatation, may also aid us considerably. The other physical signs which are present are a weak, diffuse impulse and weak cardiac sounds.

 2. The *Constitutional Symptoms* may be so few and slight that at the time they may pass almost unnoticed. But since simple endocarditis usually complicates some other disease (*e.g.*, acute rheumatism), the constitutional symptoms largely depend upon the severity or mildness of the primary disease. The onset of the endocarditis in these circumstances may be suspected when there is a sudden increase in the rapidity of the heart, and a further rise of temperature without apparent cause. Palpitation may be present, but pain and distress about the praecordium are generally absent—a feature worth bearing in mind. In the rare instances in which acute endocarditis occurs primarily, the temperature is irregularly intermittent (100° to 102° F.). The presence of such a pyrexia, and the absence of physical signs, excepting those referable to the heart, are the only data upon which we can rely for the *diagnosis* of the disease.

3. *Emboli* do not usually occur, at any rate, until very late, in *simple* endocarditis attacking a heart previously healthy. But when it attacks a heart the seat of old valvular mischief—known sometimes as RECURRENT ENDOCARDITIS—emboli may arise in various situations from the separation of the inflammatory material on the valves, and the temperature may vary from 100° to 102° F. for days, weeks, or even months. Emboli may arise from two sources: (1) Vegetations or fibrin from an inflamed valve; (2) Portions of a thrombus from the clotted blood in a dilated chamber, resulting from endocarditis and myocardial weakening. Rigors, with tenderness and enlargement of the spleen, may indicate embolism of that organ; sudden hemiplegia or other nerve troubles may point to embolism in the brain; sudden occurrence of bloody albuminous urine, with a rigor, points to embolism in the kidney; sudden blindness, to embolism of the central artery of the retina; sudden pain and tenderness

in a leg or arm may indicate plugging of one of the arteries, in which case the pulsation will be absent below the blockage; and sudden abdominal pain with vomiting and collapse may follow embolism of the mesentery.

Causes of Benign Endocarditis.—A history, or evidence at the time, of the causes of endocarditis may help us in the diagnosis. (i.) Undoubtedly the most common of these is rheumatic fever, old or recent, and it should be remembered that acute endocarditis may arise quite early in the course of the disease, before the joint lesions are manifest. Exposure to cold is mentioned as a cause, but the endocarditis in such cases is probably of a rheumatic kind. (ii.) Chorea, scarlatina, typhoid, and many other bacterial infections, may give rise to endocarditis. (iii.) It is also an occasional complication of syphilitic,[1] cancerous, and other cachectic conditions, chronic alcoholism and renal disease. (iv.) Valves deformed by acute or chronic endocarditis are predisposed to acute inflammation, and the recurrent endocarditis above referred to thus arises. (v.) The patient is generally young, usually a child, rarely older than thirty-five or forty when attacked by endocarditis for the first time.

The *Diagnosis* of benign endocarditis has been referred to above (under the constitutional symptoms), and it is not usually difficult. It is most important, however, to distinguish the two forms of endocarditis, as they differ so widely in their duration and fatality. Malignant or ulcerative endocarditis differs clinically (1) in the greater severity of the constitutional symptoms, which may present all the features of septicæmia or of the typhoid state ; (2) in the wide range of the temperature in the course of twelve or twenty-four hours, and the occurrence of severe rigors and sweats ; (3) in the invariable occurrence of systemic emboli, which may be of an infective character. When, however, malignant endocarditis supervenes on a previously damaged heart the diagnosis may become extremely difficult.

The *Prognosis* of simple endocarditis, though the malady may last for many weeks, or even months, is favourable as regards life, but the damage to the cardiac valves is generally permanent, and then the prognosis turns on many important considerations (§ 55).

Treatment should be directed primarily to the disease of which endocarditis is a complication—salicylate of soda, in sufficient quantity, for instance, for rheumatic fever, though this drug is usually thought to have no control over the cardiac lesion. *Perfect rest*—hardly allowing the patient to turn in bed—is absolutely essential. This not only favours the subsidence of the inflammation, but prevents the violence of cardiac action, which separates the fragments from the valves and leads to embolism. Tincture of aconite in ℳi doses is of value to slow and steady the heart. In this and other respects the treatment is much like that of

[1] H. L., a lad aged fifteen, was admitted into the Croydon Hospital in 1882 with intense anæmia, intermitting pyrexia, and a loud endocardial murmur. The cause of his illness was obscure during life, but he died gradually of asthenia. After death gummata were found involving the cranial and other bones. There were evidences of syphilis and of acute recent endocarditis and a generalised endarteritis.

pericarditis (§ 42), though the local treatment has less effect in endocarditis. Stimulants and digitalis are indicated only if the heart's action is weak, and they should be given with great caution, for fear of stimulating the heart too much and promoting embolism.

§ 46. **Ulcerative or Malignant Endocarditis** (Synonym—Infective Endocarditis). —In this form the endocardium is much more seriously affected, for there is more destruction of the valves and adjoining surfaces, so that large ulcers may be produced, and the valves may be perforated, or even completely disappear. The vegetations, too, are much larger.

It seems doubtful if the disease ever occurs as a primary affection, but it is rather a septicæmia or blood infection, in which the heart valves form a nidus for the circu-
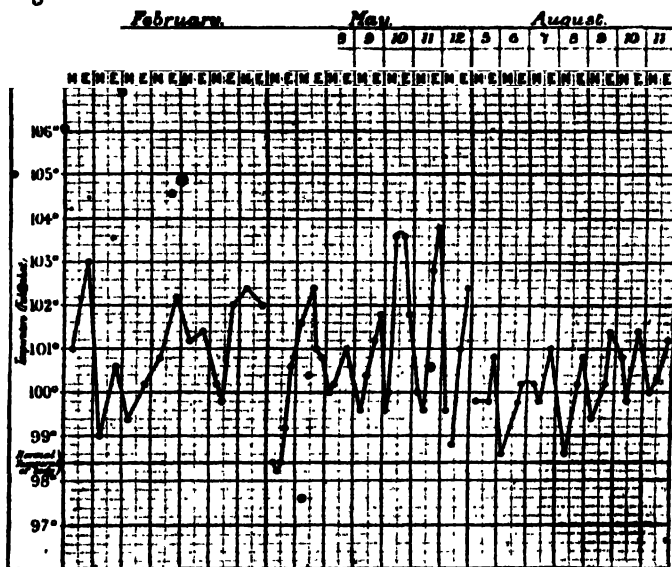
FIG. 15.—Chart of Malignant or Ulcerative Endocarditis.

lating organisms. The micro-organisms most commonly found are staphylococci and streptococci, pneumococci, and, more rarely, gonococci, bacilli coli communes, typhoid and influenza bacilli. It is, therefore, usually a complication of such diseases as influenza, pneumonia, erysipelas, septic wounds, abscesses, meningitis, gonorrhœa, dysentery, or puerperal fever. It is met with more rarely after chorea, scarlatina, and rheumatism, diseases in which simple endocarditis is so common; and with extreme rarity after tuberculosis, diphtheria, and variola. There is a marked predisposition for the disease to attack a heart which is already the seat of chronic endocarditis. (1) The *Constitutional Symptoms* vary considerably, but are usually grave, comprising intense anæmia, great prostration, and, in (a) the *Typhoid variety*, the early supervention of somnolence and muttering delirium. In (b) the *Septic variety* (such as arises with acute necrosis, the puerperium, or an external wound) the mind remains quite clear to the end, but rigors and sweats are prominent, simulating malaria, or pyæmia. The spleen is usually enlarged, and petechial rashes are fairly frequent. The fever is high, and may be continuous, but it more often runs an irregularly intermittent course, which may extend over weeks or months. (2) Generally there is a

Cardiac Murmur, but a careful examination may be necessary, and occasionally—in cases free from old valvular mischief—there is none ; the occurrence of embolism may be the first symptom to draw attention to the heart. Alteration in the character of the murmur and fresh murmurs appearing during the course of the illness afford evidence of the nature of the endocarditis. (3) The *Emboli* may be simple blockings of an artery, as in simple endocarditis, but they may become abscesses, which in turn form sources of septic infection in the lung and elsewhere (see § 43 and § 287 II). Emboli of the cutaneous vessels, producing a purpuric eruption, are not uncommon.

Course and Varieties.—The severity and duration of the disease vary widely. Those cases coming on without previous cardiac mischief usually run a rapid and acute course of five or six weeks. Varieties (*a*) and (*b*) *vide supra.* (*c*) The *Cardiac group* (Bramwell)—those in which previous chronic valvular disease exists—run a prolonged course of many months, up to a year or more ; rigors are often absent, and it may be very difficult from the symptoms to decide if malignant endocarditis is present or not. Between these extremes every grade is met with, but in the end the disease is almost always fatal. (*d*) There are certain aberrant forms marked by the predominance of such symptoms as jaundice, diarrhœa, parotitis, profuse sweatings, various eruptions, or pyrexia of a continued type. (*e*) In the *ambulant* type the patients are able to get about for months with little discomfort, but there is a constantly rapid pulse, dyspnœa on slight exertion, and an evening rise of temperature of 1 to 2 degrees.

The *Diagnosis* from Typhoid fever and Miliary Tuberculosis may be impossible during the first week. The points to which special attention should be paid are (1) evidence of a changing murmur ; (2) leucocytosis ; in typhoid fever leucopenia is the rule ; (3) blood-cultures may reveal the presence of infection, and thus support the diagnosis of ulcerative endocarditis ; (4) the Widal reaction should give reliable evidence after the ninth day ; (5) signs of general bronchitis, cyanosis and evidence of tubercle elsewhere, aid the diagnosis of miliary tuberculosis.

Treatment must be conducted on the same general lines as that of the benign variety (*q.v.*), the precautions as to rest and stimulants applying, if possible, with greater force. On theoretical grounds it would be well to administer abundance of nutriment and to try antiseptic remedies, such as quinine in full doses, antipyrin, antifebrin, sulphocarbolates, guaiacol, and the like. The recognition of the fact that malignant endocarditis is part of a septicæmia has led to more rational methods of treatment. Cases of recovery by the use of antistreptococcic serum have been reported, but this treatment has been generally disappointing, because of the large number of different organisms which may cause the disease. Vaccines promise greater success : a culture of the infecting micro-organism is obtained from the patient's blood, and from it a vaccine is prepared.

B. We now turn to those cardiac disorders in which **Pain** is the leading feature. Practically speaking, there is only one condition which is diagnosed by the character of its pain, namely, ANGINA PECTORIS. The other cardiac conditions giving rise to pain are PERICARDITIS and ACUTE DILATATION.

In PERICARDITIS the degree of pain is very variable ; and it is recognised by the other symptoms and signs fully described in § 42.

ACUTE DILATATION, when of recent origin, is commonly accompanied by præcordial pain and tenderness. It may be a contributing factor in most of the conditions which produce pain, and is the probable explanation of many of the cases of so-called "mild angina" met with in digestive and nervous disturbances.

The patient, probably a male, at or past middle life, is suddenly seized with a severe " constricting " PAIN IN THE CHEST, accompanied by a sense of suffocation— the condition is ANGINA PECTORIS.

§ 47. **Angina Pectoris** is a paroxysmal affection in which the attacks consist of severe cramp-like pain in the region of the heart, attended by a sense of suffocation and impending death. The classical and severe type of this affection is happily very rare, though milder attacks, formerly known as " pseudo-angina " but now regarded as angina of a milder grade, are not uncommon.

Symptoms.—(1) An attack comes on *quite suddenly*, often after some exertion, especially after a meal or in the cold, and consists of acute pain in the heart, which radiates down the arms, especially the left arm. The site of the pain, Mackenzie pointed out, is over the distribution of the four upper dorsal nerves, across the chest ; the skin may be hyperalgesic over this area. The face is expressive of the torture which the patient suffers, and at first is of a deadly pallor.[1] The limbs also are pale, benumbed and often covered by a clammy perspiration. The patient usually stands quite still, being afraid to move for fear of increasing the agony. The sense of suffocation, of bodily discomfort, constriction of the chest, and of impending dissolution is extreme. The attack lasts from a few minutes to one or two hours, or more, and is liable to be aggravated if the patient ventures to move from the position which he may have assumed. In a certain proportion of the cases death closes the scene. (2) The heart's action, when examined, is sometimes found to be unaltered, though palpitation may be complained of. At other times the pulse may be slow and feeble, not infrequently irregular, while electro-cardiographic examination often shows a profound alteration in the mechanism of the heart's action, *e.g.*, in the intervals between the attacks there may be a marked left-sided preponderance, and during the attack a right bundle block may occur. The pulse may be irregular, and in some cases increased in rate. There may be no murmur or physical signs of any kind referable to the heart, but more usually some form of aortic valvular mischief is present (see Etiology, below). (3) The mind remains clear throughout, so that the patient appreciates fully the horror of his position. Many cases are accompanied or succeeded by a profuse flow of urine ; others by profuse perspiration. Among the less frequent symptoms are tonic muscular spasms, convulsions, and vomiting. The limbs and other parts which were the seat of pain may afterwards feel " numbed." (4) In by far the larger number of cases the patients are of the male sex, and advanced in life. Out of 88 cases collected by Sir John Forbes, 80 were men, and 72 of these were over fifty years of age. The disease also appears to affect by preference persons among the wealthier classes of society, and those who have been possessed of unusual mental capacity.

Varieties.—1. When discoverable cardiac lesions are present, the disease is known as *Symptomatic Angina Pectoris.*

2. In *Idiopathic Angina Pectoris* no organic cause can be detected.

After death one of three conditions may be found :—(i.) The heart itself may be unhealthy as the result usually of changes in the coronary vessels ; (ii.) the heart may be apparently healthy, but disease of the aorta or peripheral vessels may be present, resulting in a rise of blood-pressure. In other cases the condition is probably due to sudden vaso-motor changes producing sudden increase in the peripheral resistance to the blood flow. (iii.) The heart and the vessels may both be diseased.

Diagnosis.—(1) It is important to distinguish mild angina from severe angina. The features of mild angina are : (i.) The attacks come on at any time of life, whereas

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[1] This pallor of the surface is generally succeeded by a reddish, or sometimes cyanotic, tint of the same parts, as I have several times observed in patients at the Infirmary during the attacks. [Trousseau (Clin. Lect. New Syd. Soc., vol. iii.) and Anstie (Trans. Clin. Soc., vol. iii.) have also noted this stage.] The succeeding stage of cyanosis is due to the paralytic dilatation which sometimes follows the spasm of the arterioles.

severe angina is confined to persons of the male sex over forty-five years of age ; (ii.) it may occur in either sex, the hysterical form being specially liable to affect young women ; (iii.) it may come on spontaneously, without previous exertion (though this is not constant) ; (iv.) the attacks often appear after meals, and are nearly always associated with some gastric derangement, such as dilated stomach, flatulence ; (v.) the pulse is usually rapid and regular, never slow ; and the sounds and boundaries of the heart are normal. Nevertheless, mild angina may or may not be associated with cardiac lesions. (2) *Acute Œdema of the Lung* is not infrequently mistaken for angina. The patient suddenly experiences a severe sense of constriction and suffocation associated with cough and frothy expectoration (§ 97). (3) *Biliary Colic* has occasionally to be diagnosed from angina, but here the patient advanced in years is usually of the female sex, and the condition is speedily followed by jaundice. (4) The diagnosis from the other causes of præcordial pain has already been given (§ 23). .

Etiology.—The clinical and anatomical antecedents of angina are the following : (1) Fatty, or fibroid, or granular degeneration of the heart muscle is said to be the most frequent. (2) Aortic valvular disease ; mitral disease is rare. (3) Advanced atheroma or calcification of the aorta. (4) Aneurysm or dilatation of the aorta, especially of the root within the pericardial sac. (5) Atheroma of the coronary arteries, calcification, or some other disease of these structures ; and this may in some cases lead to embolism or thrombosis, and thus to a more or less localised degeneration of the cardiac muscle (Kernig). (6) Arterial sclerosis (using that term in its widest sense to indicate any thickening and rigidity of the arterial walls). (7) Gumma of the heart wall, in which circumstances the patient may be young.   .

The immediate cause of an attack is usually some undue exertion. Severe Angina Pectoris is due to a *sudden* demand for increased effort on the part of a *spendthrift heart.* In, at any rate, a certain proportion of cases this sudden demand consists of an abrupt increase in the peripheral resistance by *contraction of the peripheral arterioles.* It appears that for the production of the attacks of angina the combination of these two factors is necessary. Neither of these can alone produce a paroxysm ; for, as Broadbent pointed out, high blood-pressure is extremely common, alone ; so also is a degenerated heart wall—yet angina is rare. When, however, the two are present in combination, a third or determining cause (*e.g.,* some unusual exertion, or a further increase in the blood-pressure), supervening *suddenly,* may produce an attack of angina.

Prognosis.—Severe angina is an extremely serious condition. The patient may die in a paroxysm. The attacks are sure to return, though this may not happen for some years. When there is no marked arterio-sclerosis and there is response to treatment, the outlook is good. In women who have had long atrain, as in nursing sick relatives, · both mental and physical exertion must be prohibited, and in such cases complete recovery is usual. The existence of a cardio-valvular lesion does not materially modify the prognosis ; the condition of the cardiac wall is our best guide to the probable course of a case (§ 55).   ↔

Treatment.—(a) *For the Attacks.*—Amyl nitrite, ℳ 3 to 5 ℩(0·2–0·3), inhaled, generally gives prompt relief, a method of treatment for which we are indebted to Sir Lauder Brunton. Sufferers should carry about with them glass capsules containing this quantity, which can be broken into the handkerchief.[1] The remedy hastens the advent of the second stage of arterial dilatation, and the attack passes off. For a more lasting effect, nitroglycerine may be given internally, ℳ ½ (0·06) of 1 per cent. solution every one to four hours, in tabloids, pushed to tolerance to ℳ x (0·6). All the nitrites have a similar action in dilating the peripheral arteries ; and lately advantages have been claimed for erythroltetranitrate, in that its effects are more permanent ; 1 grain administered in 1 drachm of absolute alcohol, suitably diluted, being said to have effects lasting four to five hours. It is reported to have relieved cases in which other remedies have failed. A hypodermic injection of a full dose of morphia, if the

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[1] This remedy seems to lose its effect when preserved in the ordinary way in a bottle.   .   ·   ·   ●

last-named remedies are not at hand, generally gives some relief; and in very severe cases chloroform, einhaled to complete anæsthesia, has been recommended. If this be combined with a dose of morphia, its effects become more prolonged. Hot drinks and large doses of oxygen are useful. In some cases of mild angina warm baths give great relief. I have not tried this treatment in cases of true angina, but, judging from the good effects I have observed in other conditions of vascular spasm, this method of treatment would be very efficacious in cases where movement is not harmful to the patient. Mustard plasters and warm fomentations to the epigastrium may be tried, either during or between the attacks. In two cases of mild angina with frequent acute pain I found that the following draught, carried by the sufferer in his pocket; and taken at the outset of an attack, was attended by prompt relief : Tinct. lobeliæ æth. ; spiritus etheris ; liquoris morphinæ, āā. ℥ xx. (1·2); aquæ chloroformi, ℥i (30). Stretching of the pectoral muscles by powerful movement against resistance, e.g. raising the body with outstretched arms while grasping the bed rail has been strongly advocated as a means of affording rapid and lasting relief from an attack of anginal pain.

(b) *Between the Attacks.*—It follows from the above remarks on the etiology that the indications for treatment lie in two directions—to relieve high blood-pressure or any tendency to vascular spasm, and, if possible, to restore the damaged heart. If the main element of the case is cardiac enfeeblement, this should receive our special attention, on the lines mentioned elsewhere (see § 56 and § 61). If, on the other hand, the peripheral resistance is excessive, our treatment should be directed to reduce it. The pulse should be examined many times, and under different conditions, during the day, and if the blood-pressure is very high, much may be done, even though the arteries be diseased. Erythrol-tetranitrate and nitroglycerine are here again valuable remedies, not only to relieve, but to prevent the occurrence of the attacks, and these may be combined with various cardiac tonics, such as iron, nux vomica, and especially arsenic. Digitalis and stimulants may be administered on the same principles as in cardiac valvular disease. To insure rest at night chloral, gr. v., may be given. Bleeding often affords immediate relief, and recently section of either the second, third or fourth dorsal nerve roots or of the depressor nerve has been advocated. Much may be done by regulating the mode of life, and avoiding those things which are known by experience to induce the seizures. Repose of mind and body must be strictly enforced. Other determining causes met with are exposure to cold, indigestion, dilatation of the stomach by too heavy meals, and sudden alterations of posture. Such conditions must be avoided, as also any unnecessary or sudden exertion or emotion.

C. § 48.   We now consider those conditions in which examination reveals **Enlargement** of the **Area of Præcordial Dulness.**

A. If there is a history of acute onset, and there is PYREXIA, the condition is due to ACUTE PERICARDITIS, which is fully described in Group I, § 42, or to DILATATION secondary to a febrile process.

B. If there is no Pyrexia, the enlargement may be due to

I. Cardiac Hypertrophy.
II. Cardiac Dilatation.
III. Hydropericardium.
IV. Adherent Pericardium.
V. Congenital heart disease (rare).

Aortic Aneurysm and Mediastinal Tumours must be remembered, because their existence is often revealed by finding enlargement of the præcordial dulness, or dulness above, merging into that of the heart.

Chronic conditions which may be, but are NOT NECESSARILY, attended

by ENLARGEMENT of the area of præcordial dulness should be borne in mind; their diagnosis may depend mainly on auscultation, and hence they are described under Groups D and E. .

Method of Procedure.—It will be remembered that the routine examination of the heart consisted of (1) inspection; (2) palpation; (3) percussion of the præcordial dulness; and (4) auscultation. The student should bear in mind the various *fallacies* which may give a false impression of cardiac enlargement, and also those conditions, such as emphysema, which obscure an enlarged heart (§ 35).

I. *The* APEX BEATS BELOW *its normal position; the impulse is* FORCIBLE *and heaving; on auscultation, the first sound is* DULL *and prolonged. There is* HYPERTROPHY OF THE HEART.

§ 49. **Hypertrophy of the Heart,** and the dilatation which not infrequently accompanies or follows it are certainly the commonest conditions which produce an increased area of præcordial dulness.

Cardiac Hypertrophy is an increase of the muscular substance of the heart, and its weight, which is normally about 8½ ounces in women and 9½ ounces in men, may be increased to 10 or 12 ounces, and on rare occasions to 15 or 20 ounces. Its *signs* are as follows: (1) The increase in the præcordial dulness is downwards and outwards if the left ventricle be hypertrophied, outwards if the right ventricle; (2) the apex beats below its normal position; (3) the impulse is unduly forcible, heaving, or thrusting; (4) on auscultation, the first sound is muffled, less audible, and prolonged. The pulse is firm, strong and bounding.

Symptoms may be altogether wanting if the hypertrophy accurately compensates for the obstruction in the circulation which has caused the hypertrophy. The patient may, indeed, be unaware of any cardiac disorder. But generally, on inquiry, he will complain of a "thumping" in his chest and "throbbing" in his head, occasionally of breathlessness and præcordial distress.

Etiology.—Hypertrophy, with the dilatation usually accompanying it, is the result of some obstruction in the circulation, either in the lungs (*e.g.*, bronchitis with emphysema) or in the general circulation (*e.g.*, cardiac valvular disease, arterial thickening or high blood-pressure as in chronic renal disease). It is an illustration of the physiological law that increased use leads to increased growth. The part of the heart which chiefly undergoes hypertrophy will depend on the position of the obstruction; and the signs met with in addition to those above mentioned will vary accordingly. Thus, there will be three sets of symptoms: (*a*) Signs of hypertrophy of the heart; (*b*) signs of enlargement of the cavity specially involved; and (*c*) signs and symptoms of the cause. The following causes will be more readily understood by consulting Fig. 16 (p. 80), and it must be remembered that the enlargement is never in actual practice strictly limited to one chamber of the heart.

(a) HYPERTROPHY OF THE LEFT VENTRICLE is indicated by enlargement of the area of dulness, chiefly towards the left ; the apex beats *below* and to the left of its normal position ; the pulse is strong unless modified by the presence of a valvular lesion, and the carotids may be seen to pulsate.   This condition may arise from eight different causes :

(i.) *Mitral regurgitation,* in which case there would be a systolic apical murmur, and the other features given in § 53.

(ii.) *Aortic stenosis* or *regurgitation,* which may be recognised by a basal murmur of systolic or diastolic rhythm respectively, and other characters given in § 53.   The hypertrophy resulting from regurgitation may be greater than that due to any other cause (*cor bovinum* of the older writers).   The heart may weigh in these cases 20 to 30 ounces, or more.   In regurgitant lesions a certain amount of dilatation always accompanies hypertrophy, and the condition is then known as " eccentric " hypertrophy.   In these cases the dilatation is compensatory, and produces no untoward symptoms.   True, or, as it is called, " concentric," hypertrophy, unaccompanied by any dilatation, is only met with in aortic stenosis and Bright's disease.

(iii.) *Aneurysm of the aortic arch,* if unattended by valvular disease or renal mischief, does not *per se* cause cardiac hypertrophy ; but it is nearly always so attended, and thus becomes a fairly frequent cause of hypertrophy of the left ventricle.   If the aneurysm involves the first half of the arch, it produces marked physical signs ; if the second or third part, *pressure* symptoms arise without signs (§ 65).

(iv.) Prolonged *high blood-pressure*—and thus its numerous causes (§ 72)—may lead to hypertrophy of the left ventricle.   It is probably in this way that chronic Bright's disease is so frequently accompanied by hypertrophy of the left ventricle.

(v.) Widespread *thickening of the peripheral arterioles* is invariably followed sooner or later by a certain degree of hypertrophy of the left ventricle (arterial sclerosis, § 78).

(vi.) *Excessive muscular exercise,* whether athletic or laborious, may produce hypertrophy, and in support of this statement it may be mentioned that the normal increase with age is more noticeable in men than in women.

(vii.) *Increased density of the blood* is a cause of hypertrophy which is not mentioned among the lists usually given in textbooks, but which, nevertheless, must be of considerable potency.   It is estimated that 99 per cent. of the work done by the heart is employed in overcoming the resistance due to capillarity in the arteries and capillaries, and it follows, almost of necessity, that if the density of their fluid contents be increased, the resistance will be increased proportionately.

Obscure Causes.—If in a given case of cardiac hypertrophy careful examination reveals no valvular mischief, and no obvious cause can be made out, the physician should always suspect OBSCURE AORTIC ANEURYSM, OBSCURE RENAL DISEASE, or widespread ARTERIAL THICKENING.

(b) HYPERTROPHY OF THE RIGHT VENTRICLE is indicated by enlargement of the area of dulness to the right ; throbbing and pulsation in the lower right intercostal spaces and epigastrium.   It is the result of resistance to the emptying of the ventricle into the pulmonary vessels.   This may occur in

(i.) *Pulmonary diseases* attended by obstruction in the pulmonary circulation, of which *bronchitis with emphysema* is certainly the most frequent.   This condition, a very common one, is identified by a history or evidence of lung mischief (§ 120).

(ii.) *Mitral stenosis* is the next most common cause, and should be borne in mind even in the absence of a presystolic murmur (§ 54).

(iii.) *Mitral Regurgitation.*

In (i.) the total vascular area is decreased by the lung condition ; in

(ii.) and (iii.) the intra-pulmonary blood-pressure is raised and the vessels are already over-filled.

(c) HYPERTROPHY OF THE LEFT AURICLE is always attended by dilatation. It is a difficult condition to recognise with certainty, because palpable and visible pulsation in the third left interspace, when present, though often due to this cause, may admit of other explanations.

It may arise in *mitral regurgitation*, but its chief cause is *mitral stenosis*. In the latter condition, palpation generally reveals a thrill over the apex, and careful auscultation may detect the presystolic or mid-diastolic murmur (§ 53).

(d) HYPERTROPHY OF THE RIGHT AURICLE gives rise to the following physical signs : (i.) Increase of dulness to the right of the sternum ; (ii.) powerful jugular pulsation, which polygraphic records prove to be due to forcible auricular contractions

(e) Extreme HYPERTROPHY OF BOTH AURICLES AND VENTRICLES arises in congenital heart disease, but may be confined to the right side. It also occurs with Adherent Pericardium of the external type (§ 51).

Prognosis and Treatment.—Cardiac Hypertrophy is in itself essentially a conservative process for some condition which causes obstruction in the circulation. It is Nature's method of compensating for the obstruction, and it is well to promote it up to a certain point.

1. *If the cause be removable,* the prognosis is favourable. Our treatment in such cases should therefore be directed to the removal of the cause—*e.g.,* high blood-pressure, which can be reduced.

2. *If the cause be not removable,* the prognosis of the case depends on our being able first to maintain the compensatory hypertrophy, and, secondly, to relieve the heart of part of its work, so that dilatation does not supervene on the hypertrophy. To accomplish the first, we should endeavour to promote the general nutrition by tonics and hygienic measures, and by regulating the bowels with mild saline laxatives, and similar remedies. In order to relieve the heart of part of its work, and to aid the systemic circulation, baths, massage, passive and active movements are of the greatest use (see § 56).

3. The *existence of cardiac hypertrophy* indicates an element of risk to a person's life from one of three factors. In the first place, hypertrophy nearly always indicates that there is obstruction somewhere in the circulation, and this, whatever it be, is in itself an injury to health, and may shorten life. Secondly, a far more important consideration is the increased liability to cerebral hæmorrhage and vascular rupture elsewhere. Thirdly, high blood-pressure is an almost invariable accompaniment of cardiac hypertrophy, and this causes a continual strain upon the peripheral vessels, which results first in arterial hypermyotrophy and later in arterial sclerosis, the serious consequences of which are indicated elsewhere (§ 78).

II. *The* AREA OF DULNESS IS INCREASED ; *the position of the* APEX-BEAT IS INDEFINITE ; *the impulse is diffuse and wavy ; on auscultation, the first sound is short and sharp. The condition is* CARDIAC DILATATION.

§ **50. Cardiac Dilatation** (one form of " Cardiac failure ") is an indication that the heart is " failing " to keep pace with the extra demand made upon it, that the reserve power of the muscle wall is becoming spent. Dilatation is the immediate physiological response of the heart to increased work. If increased work continues, hypertrophy normally follows. Physiological dilatation is limited by the pericardium. In some diseases dilatation is found although the work of the heart is diminished as much as possible by keeping the patient in bed. This is considered to be due to a change in the state of the muscle cells in the heart wall. In febrile conditions it is extremely common for some degree of cardiac dilatation to occur. This is especially frequent in Acute Rheumatism, and may occur without any evidence of endocarditis. A true rheumatic myocarditis with definite foci of cell infiltration has, indeed, been shown to occur ; the most suggestive clinical indication of the condition is a mitral systolic bruit, due to regurgitation through a dilated mitral ring.

The *Physical Signs* of cardiac dilatation resemble those of hypertrophy in several ways ; in both the area of dulness is increased chiefly in a transverse direction, to the right or to the left, according to the chamber which is dilated. But there are two important features specially belonging to dilatation : (1) The cardiac impulse is wavy and diffuse, and is displaced outwards rather than downwards ; it may be so feeble as to be hardly perceptible. (2) On auscultation, the first sound at the apex is clear and sharp, resembling the normal second sound in character. Murmurs may be present from co-existing valvular disease, but *a systolic murmur*—the " murmur of dilatation "—may sometimes be heard apart from actual valvular disease, because the auriculo-ventricular orifices, by reason of the dilatation, allow a reflux of the blood. The pulse may be feeble, rapid and sometimes irregular.

TABLE III.—DIAGNOSIS OF TYPICAL CARDIAC HYPERTROPHY FROM
TYPICAL DILATATION

	Apex-Beat and Impulse : Displaced in Both.	Percussion.	Auscultation.	General Symptoms.
Hypertrophy.	Forcible, heaving, thrusting; below and to l. of normal (l. ventricle) ; pulsation in epigastrium (r. ventricle).	L. Chambers : Area increased transversely to the l. R. Chambers : Area increased transversely to the l. and r.	Sounds muffled, prolonged, and forcible.	May be absent ; or symptoms of high blood pressure.
Dilatation.	Feeble, undulatory, diffuse. If r. ventricle—pulsation in the epigastrium.	L. Chambers : Area increased transversely to the l. R. Chambers : Area increased transversely to the r. and l.	Systolic murmur at apex, at one stage. Systolic murmur in tricuspid area, at one stage. Sounds sharp and clear.	Dyspnœa, cough, cyanosis, and other signs of lung congestion. Dropsy, scanty high-coloured albuminous urine, enlarged liver, ascites, and other signs of congestion of organs.

Towards the end, when cardiac failure is extreme, fœtal rhythm, gallop rhythm, and " delirium cordis ".may occur. In fœtal rhythm the long and short pauses are almost idéntical, so that the first and second sounds can scarcely be distinguished; in gallop rhythm there is rapidity of action, together with a distinctly reduplicated second sound. In delirium cordis the heart is so rapid and so irregular that it is practically impossible to make out the relations of sounds and murmurs.

It is, however, by the presence of certain *symptoms* that the existence of cardiac dilatation (or failure of compensation) generally becomes manifest. In hypertrophy, as we have seen, there may be no symptoms at all; but with dilatation the patient complains of : (1) *Heart symptoms*, such as breathlessness on little or no exertion, palpitation, and præcordial distress. (2) There may also arise a number of *symptoms referable to other parts*, in consequence of the delay in the circulation; such as anasarca, ascites, and symptoms of congestion of the lungs, liver, and kidneys. These will be described under Cardiac Valvular Disease, where the means of detecting which cavity is chiefly involved are also given (see also Table III).

The *Causes of Cardiac Dilatation* are of extreme importance as bearing on the prognosis and treatment of cardiac valvular disease and other circulatory disorders. The *clinical conditions* which produce dilatation are practically identical with those which produce cardiac hypertrophy (§ 49), when they are persistent and *are associated with some condition which impairs the nutrition of the heart* (see (b) below). Undoubtedly the two commonest causes of cardiac hypertrophy and dilatation are CARDIAC VALVULAR DISEASE and CHRONIC BRONCHITIS WITH EMPHYSEMA, and these are the possibilities which should first suggest themselves to the mind in a case where dilatation is evident. The former is fully discussed in § 53.

Emphysema, with its usual accompaniment of Chronic Bronchitis, produces in time a dilated *Right Ventricle*. This latter is recognised by two very characteristic local signs, in addition to the breathlessness, etc., above mentioned—viz. : (i.) epigastric pulsation; and (ii.) pulsation in the jugular veins. The clinical picture presented by this frequent pathological combination is very characteristic—the florid face and plethoric build ; the easily excited breathlessness and constantly recurring cough, enable us to recognise the condition almost at a glance. The subject is more fully discussed under Cardiac Valvular Disease (§ 53).

The *essential or pathological causes* of Dilatation may be arranged under four headings :

(a) Any condition which persistently *prevents the complete emptying of the cavities of the heart* (see Causes of Hypertrophy), will produce compensatory hypertrophy with dilatation, which will be *exactly proportional* to the increased resistance in the circulation, provided none of the circumstances mentioned under (b), (c), or (d) below are also present. If any of these circumstances are in operation, dilatation or failure may be initiated without previous or accompanying hypertrophy. Moreover, the supervention of any of these in the course of a cardiac case may at once disturb a well-balanced compensatory hypertrophy, and serious symptoms may immediately appear.

(b) Any *failure of general nutrition*, or vitality, may entail a weakened cardiac wall,

which will perhaps yield even under normal circulatory conditions.   Such, for instance,
may be caused by exposure, insufficient food, alcoholic excesses, old age, various fevers
(especially rheumatic fever, typhus, typhoid, and malaria), various toxæmic conditions
(such as pernicious anæmia, scurvy, chlorosis, coli and streptococcal infections), and
cachectic conditions (such as syphilis, tubercle, and cancer). See Causes of Pyrexia
and of Anæmia (Chapters XV and XVI).

(c) *Local impairment of the nutrition of the heart wall* may result in dilatation without
hypertrophy, even with normal circulatory resistance.   Myocarditis, for instance, and
the conditions which accompany peri- and endo-carditis (which lead sometimes to
acute dilatation) ; or the more gradual degenerations which ensue on sclerosis and
other diseases of the coronary arteries ; or fibroid and other degeneration of the cardiac
wall (§ 52), .Any of these may upset the balance of a well-adjusted hypertrophy.

(d) Any *sudden strain on an apparently normal heart* may produce acute dilatation.
Thus, severe and sudden grief, fright, or anxiety may damage the heart through its
nervous apparatus, and severe muscular exertion in athletes or soldiers who have not
had any previous training may cause the heart to give way and dilate.   Instances of
the latter are met with in hill-climbers who are " out of form," and others who take
sudden and unaccustomed exercise.   But it is probable that some pre-existing condi-
tion such as infection, etc., is present.   Breathlessness may date from incidents of
this kind, from which the patient may never, or only with difficulty, recover.   Rest
and gentle exercise are indicated.   Prolonged fatigue may also act locally by overtaxing
the heart muscle.   •

The *Prognosis* and *Treatment* of Cardiac Dilatation are fully dealt with under
Cardiac Valvular Disease (§ 53).

III. *The area of dulness is* INCREASED UPWARDS, *and its shape is pyra-*
midal, with the point upwards ; the apex-beat is raised, and the impulse is
weak and undulatory ; on auscultation, the sounds are feeble.   The disease
is HYDROPERICARDIUM.   •

§ 51. Hydropericardium is a chronic effusion of fluid into the pericardium. (1) The
shape of the dulness is very characteristic, being pyramidal, with the narrow end
upwards.   (2) The apex of the heart is *raised*, and *to the right* of its normal position,
because the roof of the pericardium is raised by the fluid, and takes the heart with it.
(3) For the same reason, the left margin of præcordial dulness extends *beyond* the apex-
beat. (3) On auscultation, the heart sounds are distant and muffled.   There may be
.irregularity and rapidity of the pulse, and difficulty of breathing from the impeded
action of the heart and lungs.

Etiology.—Chronic effusion into the pericardium may originate in one of three ways.
(1) As the result of Acute Pericarditis (§ 42), of which a history is generally obtainable,
but by no means always (see Latent Pericarditis, § 44).   Most authors draw a dis-
tinction between chronic pericardial effusion of inflammatory origin and simple dropsy
of the pericardium (hydropericardium proper).   But the physical signs are practically
indistinguishable ; for the diagnosis of the former we depend mainly on the history
of it having commenced as an acute affection, and on the absence of anasarca.   (2)
True hydropericardium seldom occurs excepting as part of a general dropsy due to
renal or cardiac disease, and therefore the urine should be carefully examined.   In
these circumstances dyspnœa is the most obvious symptom complained of and an
X-ray examination should always be made if there is any doubt as to the existence
of fluid.   (3) If hydropericardium be not preceded by pericarditis, or be not part of
a general dropsy, new growth or tubercle, although rare, should always be suspected.
In these circumstances, if a little fluid be withdrawn by a hypodermic syringe (§ 42),
it may be blood-stained (cancer) or contain bacilli (tubercle).

The *Diagnosis* from Cardiac Dilatation should be readily accomplished by the
shape of the dulness, which is square instead of pyramidal in dilatation ; and by
the heart sounds, which are clear and sharp in dilatation, muffled in effusion.

X-ray examination is of assistance. Pleuritic effusion is attended by pulmonary symptoms.

The *Prognosis* of hydropericardium depends on its causation, being favourable in Cause 1, adding only a little to the gravity of the primary malady in 2, and being almost necessarily fatal in 3.

Treatment.—The treatment of inflammatory effusion is dealt with in § 43. If part of a general dropsy, our efforts must be directed to this. Counter-irritants are sometimes useful. Paracentesis should not be considered unless the cardiac embarrassment is very urgent, because of the danger of withdrawing a large amount of fluid suddenly from the pericardial sac.

IV. **Adherent Pericardium** may exist in two forms : (i.) the *internal*, in which the visceral and parietal layers are adherent in varying degree. The condition is not recognisable during life and leads to no symptoms. The heart can act as well as if a pericardial space were present (§ 44). (ii.) *External* adhesions may bind the pericardium to all the surrounding structures. The condition is a sequel to pericardial effusion, with softening and dilatation of the sac. In many cases some degree of internal adhesion is also present. The condition is serious, and leads to extreme cardiac embarrassment ; the heart is dilated, and is so tethered that it cannot return to its normal size. The signs are many, but not very reliable. They are (1) a systolic tug at the apex ; (2) fixity of the cardiac apex during respiration and with change of position ; (3) systolic recession along the attachments of the diaphragm, either in front along the lower costal border, or behind under the eleventh and twelfth ribs ; (4) signs of hypertrophy, greater than can be accounted for by the severity of any valve disease which may be present ; (5) pulsus paradoxus, or diminution or disappearance of the radial pulse during inspiration of normal depth ; (6) signs of incompetence of auriculoventricular valves ; (7) a diastolic shock in the veins of the neck. The subjects of this condition seem unable to acquire more than a slight degree of " compensation " ; slight improvement is soon followed by more complete exhaustion of their " heart-force." No permanent improvement is to be expected. Sudden death is common. Cardiolysis or removal of ribs has been successfully performed for this condition.

V. In CONGENITAL HEART DISEASE the enlarged area of præcordial dulness is distorted and somewhat square. There is a characteristic murmur. For the differential signs of this condition, see § 54.

VI. In ANEURYSM of the first part of the aortic arch, the upper part of the dull area is increased transversely, and there is dulness over the sternum. Auscultation reveals a diastolic murmur and a loud, sharp second sound at the base of the heart (see § 65).

D. We now turn to those *conditions in which there is found an alteration of the heart sounds, or a murmur.*

It is well to bear in mind several fallacies referred to on p. 50.

In the absence of these, if *the heart sounds are* FAINT, the disease is MYOCARDIAL DEGENERATION.

The second sound is ACCENTUATED *or* DOUBLE. This may be due to (i.) High blood-pressure ; (ii.) aortic aneurysm ; (iii.) pulmonary stasis in mitral disease.

The first sound at the apex is unduly LOUD. This may be due to : (i.) Mitral stenosis ; (ii.) thin chest wall ; (iii.) nervousness of patient ; (iv.) hypertrophy of the ventricle ; (v.) adjacent air-containing cavity acting as a resonator.

The first sound is unduly SHORT. This may be due to : (i.) Dilatation

of the ventricles ; (ii.) rapidity of the pulse ; (iii.) feeble contraction of the 'left ventricle (mitral stenosis) secondary to incomplete distension which results in inadequate stimulation ; (iv.) myocardial degeneration ; (v.) myocardial toxæmia.

I. *On auscultation the* HEART SOUNDS ARE VERY FEEBLE ; *the impulse is weak. No murmur is heard.* MYOCARDIAL DEGENERATION *may be strongly suspected.*

§ 52. Myocardial Degeneration.—Under this term are included Fatty Heart and Fibroid Heart.

• The *signs* and *symptoms* fall into three groups : (1) Failure of tonicity, causing dilatation ; (2) failure of contractility, causing circulatory inadequacy ; (3) changes in the primitive musculature, causing irregularities of rhythm (see § 57). Supposing the most careful examination reveals *no physical signs*, although, by reason of certain *subjective symptoms*, we believe the patient to be suffering from cardiac disease, MYOCARDIAL DEGENERATION OF THE CARDIAC WALL should be suspected. The diagnosis often rests on a process of exclusion, and the two forms are clinically indistinguishable from one another. Its detection is often a matter of some difficulty, but the disease may be *suspected* (i.) when the heart impulse and pulse are feeble, and the heart sounds are weak ; (ii.) if the patient be subject to attacks of fainting or dizziness, or to palpitation and breathlessness. The patient may experience no symptoms at first beyond a tight feeling across the chest on exertion ; later, he may have dyspnœa, especially at night or after movement. With the onset of cardiac dilatation the area of præcordial dulness may be increased, but it is often obscured by emphysema of the lungs, which is itself one of the causes of myocardial degeneration. Some œdema of the ankles may be present. Later on, anginoid and epileptiform attacks, or the syndrome known as Stokes-Adams' disease (§ 63), are not uncommon.

The degree of degeneration is difficult to estimate clinically. Guidance on this point may be obtained from (1) the character of the heart sounds : are they of normal intensity and duration ? (2) Estimation of the amount of exercise which can be carried out without distress, such as pain, dyspnœa, giddiness, palpitation. (3) Effect of increased work on the size of the heart. A healthy heart decreases in size with an increased pulse-rate ; an unhealthy heart dilates. (4) The amount of dilatation present. (5) The difference between the diastolic and the systolic pressure ; with advancing cardiac inefficiency this difference tends to diminish. (6) The character of the electro-cardiographic curves.

Causes.—Myocardial degeneration is a consequence of interference with the nutrition of the heart wall. This may be due to (1) changes in the blood, *e.g.*, severe anæmia ; (2) toxins, *e.g.*, diphtheria, scarlet fever, typhoid and rheumatism ; (3) changes in the coronary vessels producing narrowing of their lumen. Fatty heart is met with in two forms : fatty infiltration and fatty degeneration. The latter is of serious significance,

and is a common cause of sudden death. The disease occurs chiefly in persons past miḍdle age.

The *prognosis* is extremely grave. Fatty heart is one of the commonest causes of sudden death. The earlier stages of the malady are insidious, so that by the time pronounced symptoms appear the mischief may be irreparable. The patient may die in one of the syncopal attacks, or, if not, he will rarely live for more than six to twelve months after the appearance of definite symptoms, such as dyspnœa, Cheyne-Stokes respiration, or a continually irregular pulse, especially the pulsus alternans.. In the early stages of cardiac degeneration plenty of fresh-air exercise and good sleep are essential for increasing the reserve power of the unaffected muscle fibres, and if the patient responds to this treatment he may live for many years (Mackenzie). Prognosis and treatment are discussed more fully in § 56. Unhealthy conditions of the primitive muscular tissue are considered under irregularities of rhythm (§ 57).

D. II. *A* **Murmur** *is present.* Its source may be :
(1) Exocardial—Pulmo-pericardial, Pleuro-pericardial.
(2) Pericardial—friction of Pericarditis, " Milk-spot."
(3) Endocardial— Endocarditis of valve or wall; narrowing or dilatation of orifice of valves.; hæmic bruits ; congenital abnormalities.

The chief points to be considered in diagnosing the *source of a murmur* are given in § 37 and § 44 (Table II).

While auscultating the heart three questions should be in the physician's mind : (1) What is the character of the first sound ?　(2) What is the character of the second sound ?　(3) Is a murmur present ?

§ 53. Chronic Endocarditis—Cardiac Valvular Disease—Cardiac murmurs.

—Disease of the valves of the heart is the commonest of all cardiac disorders, and it is revealed on auscultation by the presence of a bruit or murmur, which is added to, or replaces, one or both of the heart sounds.

Method of Procedure. Five features must be carefully investigated in any murmur : TIME OF OCCURRENCE. Whether it REPLACES or merely ACCOMPANIES the first or second sound ; POSITION ; CONDUCTION ; and CHARACTER. The last named is relatively least important. In order to be quite sure of the time of a bruit, it is convenient to place the thumb on the carotid artery while auscultating the chest.

The characters of PERICARDIAL MURMURS have already been given (§ 44) ; and their diagnosis from endocardial murmurs (Table II, p. 62).

HÆMIC, or functional, MURMURS [due to loss of tone] are frequently heard in anæmia and in some other blood conditions (see Chapter XVI). They are usually systolic in time. They are rarely double. They are usually heard loudest in the pulmonary area, and are heard best when the patient is lying down. A single murmur of presystolic or diastolic time is usually an indication of organic disease at one of the cardiac orifices, but may be endocardial.

Valvular disease may be due to several lesions (§ 53), but the commonest

one in early life is endocarditis (acute or chronic), and in older persons chronic degenerative change. The effect is a thickening or puckering of the valves and ring, which.results in one or both of two conditions : (a) *Stenosis*—i.e., a narrowing (στενοω, to contract) of the orifice, which prevents the blood flowing freely through it ; or (b) *Regurgitation*, in which the valves are incompetent and allow a reflux of the blood to take place from imperfect meeting and closure of the cusps. The remote effect of these two conditions is practically the same—viz., a retardation or obstruction to the circulation of blood through that orifice.

It simplifies diagnosis very much that cardio-valvular disease arising *after birth* is practically *confined to the left side of the heart*- i.e., to the mitral and aortic orifices. Thus it happens that there are four principal valvular lesions — MITRAL REGURGITATION, MITRAL STENOSIS, AORTIC REGURGITATION, and AORTIC STENOSIS.

TABLE IV.—DIFFERENTIATION OF CARDIAC VALVULAR DISEASES

		Ausculta-tion.	Appear-ance of Patient.	Pulse.	Other Symptoms special to the Disease.
Mitral (apical murmurs)	Regurgi-tation.	Systolic murmur conducted into axilla	Florid	Often irregular in force and compressible.	Dropsy, enlarged liver and ascites, etc , with signs of congestion of organs
	Stenosis	Presystolic murmur	Patient young	Small, mode-rately firm , very irregular with onset of auricular fibrillation	Hæmopty-sis , emboli,
Aortic (basal murmurs)	Regurgi-tation	Diastolic murmur conducted down sternum.	Sallow	" Water-hammer," rapid and compressible.	Throbbing of arteries of neck, with symp-toms of cerebral anæmia and anginoid attacks.
	Stenosis [1]	Systolic murmur conducted into vessels of the neck.	Heart lesion of the aged.	Slow, regular, small and hard.	No special symptoms.

The student should study Fig. 8, p. 48, so as to thoroughly comprehend the various events which occur during one complete contraction and dilatation of the heart (a cardiac cycle). He should also bear in mind that the left side of the heart is behind the right, and that the left ventricle comes nearest to the surface only at the apex, immediately behind or just below the fifth rib (Figs. 9 and 10, pp. 49 and 51). He should also remember that a cardiac murmur is not produced in a diseased orifice, but by the eddies in the blood-stream beyond, and is conducted in the direction of the stream of blood which is causing the murmur. For these reasons a murmur is not always heard loudest directly over the orifice diseased. The student should also consult the diagram of the circulation on page 80.

Diagnosis of Cardiac Murmurs.—The first thing to determine is whether a given murmur is related to the first or second sound of the heart— i.e.,

[1] Real aortic stenosis is very rare, but atheromatous roughening is very common.

whether its time is systolic or diastolic—and this will form a convenient basis of classification of cardiac murmurs.

A. **Systolic Murmurs** [1]—*i.e.*, bruits added to or replacing the first found—may be produced by the following causes, which are mentioned more or less in order of frequency : mitral regurgitation, hæmic conditions (see above, and .Anæmia, § 432), aortic stenosis, dilatation of the root of the aorta, tricuspid regurgitation, pulmonary stenosis, congenital heart disease, and cardio-pulmonary conditions.

I. In **Mitral Regurgitation** the systolic murmur is characterised by (i.) being loudest at the apex; (ii.) being conducted to the axilla, and also audible behind, at the angle of the scapula. Owing to the resulting hypertrophy of the left ventricle, the apex is displaced downwards and outwards. There is accentuation of the second sound in the pulmonary area, due to the congestion in the pulmonary circulation. The pulse is soft, there is a characteristic florid physiognomy, and a tendency to dropsy.

The mechanical effect of Mitral Regurgitation upon the heart and lungs is as follows : (1) owing to the reflux of blood from the ventricle during ventricular systole the auricle becomes dilated. (2) In order to drive on the increased volume of its contents the auricle hypertrophies. (3) The effect of a hypertrophied auricle driving an increased volume of blood into a flaccid ventricle at each auricular systole is to produce dilatation of the ventricle. (4) When the power of the auricle begins to fail it is unable to empty itself properly, and there is

FIG. 16.—Scheme of the Circulation of the Blood.—The superior and inferior venæ cavæ (6) bring the blood back from the organs and tissues into the right auricle (1). Thence it passes into the right ventricle (2), through the pulmonary artery (7) into the lungs. Returning from the lungs by the pulmonary veins (9), it passes through the left auricle (3) and left ventricle (4), and is distributed by means of the aorta (5, 5) and the carotids (8) to the organs and tissues of the body. Notice that the blood from the stomach and intestines passes through the liver before joining the general circulation. (From Huxley's "Physiology," modified.)

[1] Systolic murmurs are sometimes spoken of as Ventricular Systolic, or V.S., murmurs, being produced by the systole of the ventricle. ●

difficulty in the free passage of blood from the pulmonary veins. Thus pulmonary blood stasis tends to occur. (5) To overcome this stasis it is necessary for the right ventricle to hypertrophy. In failing cases dilatation supervenes, often with the onset of tricuspid incompetence. (6) So that in cases of Mitral Regurgitation there may occur : (a) dilatation and hypertrophy of Left Auricle and Ventricle ; (b) Pulmonary congestion ; and (c) dilatation and hypertrophy of the right auricle and ventricle.

General Symptoms arise as soon as exhaustion of the heart muscle supervenes. They are those of blood stasis in (i.) the pulmonary vessels, and (ii.) the systemic veins. •

. 1. The symptoms of *dilatation of the left ventricle* have been already mentioned (§ 52). *Pulmonary congestion* is revealed by laboured breathing, cough, expectoration of mucus, sometimes tinged with blood, or actual hæmoptysis. The physical signs are abundant mucous râles and, sometimes, scattered patches of dulness at one or both bases. *Pulmonary infarction* may be suspected by sudden increase of dyspnœa, accompanied by hæmoptysis.

2. *Dilatation of the right ventricle*, consequent on the congestion of the pulmonary circulation, sooner or later produces the following symptoms and conditions :

(i.) A *bruit* over the tricuspid orifice is sometimes heard (see below, p. 82).

(ii.) *Pulsation* in the epigastrium.

(iii.) *Dropsy*, which indicates congestion of the whole venous system. Cardiac dropsy *starts and predominates in the legs* or *the back*, whichever may happen to have been in the most dependent position. The skin is tense, and is very liable to be attacked by erythematous, erysipelatous, and inflammatory conditions (cellulitis, ulcer, etc.). *Ascites* in varying amount is generally present. It is often an early and prominent sign in mitral stenosis. *Cyanosis* and a general lividity of the surface are consequences of the same venous stasis. A case of mitral disease, therefore, presents a marked contrast to one of aortic disease, where the countenance is pale and sallow. •·

· (iv.) *Engorgement of the liver* is evidenced by pain and tenderness in that region, and jaundice of the skin and conjunctivæ. The organ is enlarged, and it may extend even to the umbilicus. Sometimes pulsation of the liver may be made out by placing one hand on the epigastrium, and pressing the other beneath the back in the dorsal region. In cases of *dropsy with albuminuria*, when we are in doubt whether the dropsy is of renal or cardiac origin, hepatic enlargement is a valuable diagnostic aid ; its presence is very usual in cardiac cases, but it is not one of the consequences of renal disease, although the association of cirrhosis of the liver l fibrosis of the kidney is not unusual.

! (v.) *Indigestion*, want of appetite, a sense of discomfort in the stomach after meals, nausea or actual vomiting, with streaks of blood, indicate congestion of that organ.

(vi.) *Albuminuria*, with high-coloured scanty urine of high specific gravity (and possibly casts in long-standing cases), points to congestion of the kidney.

(vii.) *Splenic enlargement* and tenderness are the usual indications of congestion of that organ.

I*a*. A MURMUR OF DILATATION (Atonicity M.), systolic in rhythm, having all the above characters, and, like it, due to mitral regurgitation, may occur without definite disease of the valve, when the *left ventricle becomes dilated*, and the muscular ring around the valve *fails to complete* the closure of the mitral valve. This condition is especially apt to occur (i.) in the age when dilatation of the left ventricle supervenes on hypertrophy, and (ii.) in acute febrile conditions, notably Acute Rheumatism. It also occurs with chronic renal disease, arterio-sclerosis and anæmia.

II. **Aortic Stenosis** is another lesion producing a systolic bruit. True stenosis of the aortic orifice is very rare, but a roughness, or the presence of vegetations on the inner surface of the valves, may produce the same bruit. The latter is mostly found in old people on account of the degenerative changes, and a systolic murmur thus produced is of relatively less serious import.

True narrowing of the aortic ring should not be diagnosed unless five signs are present : (i.) a systolic bruit in the aortic area and conducted into carotids. It is harsh, sometimes musical, and may be audible at the apex. (ii.) A systolic thrill in the aortic area, second R. interspace ; (iii.) hypertrophy of the left ventricle ; (iv.) a slow-rising, well-sustained pulse ; and (v.) a weak second sound.

General Symptoms are almost wanting in aortic stenosis—other than occasional pain, pallor or sallowness of the face, and faintness or giddiness —until perhaps the mitral valve, owing to backward pressure and dilatation of the left ventricle, gives way (see Mitral Regurgitation, p. 80).

The detection of aortic stenosis is sometimes as difficult as that of mitral stenosis, and the characteristic murmur may be absent. It may then be suspected when the patient, generally an elderly man, presents persistent dyspnœa, bradycardia, nervousness, and occasionally anginoid attacks, which are not otherwise accounted for. In true stenosis the second sound is short and not very loud ; whereas in cases of high arterial pressure with systolic murmur the second sound is loud. The apex-beat in stenosis is displaced downwards.

III. In DILATATION OF THE COMMENCEMENT OF THE AORTA a systolic murmur is the most common one heard. The condition is a "relative stenosis," *i.e.*, though the aortic ring is normal in diameter it is small as compared with the diameter of the enlarged aorta. A peculiar ringing character of the aortic second sound is the most constant physical sign (§ 65).

IV. TRICUSPID REGURGITATION takes place when that orifice is diseased or DILATED. Some maintain that if the valve be healthy, though dilated, no bruit can be heard, but it is certain that in cases of confirmed bronchitis a murmur is often present which comes and goes under treatment, and which is not found to be attended with any marked changes in the tricuspid valve after death. The murmur is characterised by (i.) being heard best at the tricuspid area—*i.e.*, on the left side of the lower part of the sternum ; (ii.) it may be heard as far out as the right nipple ; (iii.) the pulse is of low tension, often irregular ; (iv.) owing to the accompanying hypertrophy or dilatation of the right auricle, the area of dulness extends to the right.

General Symptoms, as above indicated (p. 81), result from tricuspid regurgitation. By far the commonest cause is Chronic Bronchitis, which thus presents a clinical picture readily recognised.

V. PULMONARY STENOSIS is practically unknown, except as part of congenital malformation of the heart. The murmur is systolic in rhythm, loud and harsh, and is heard over a very wide area, but most distinctly in the second left interspace. It is usually accompanied by a coarse thrill.

§ 54. Congenital Heart Disease is comparatively rare. There are three cardinal signs produced by it : (1) The præcordial dulness is considerably *increased,* the normal shape is *distorted,* and may extend far beyond the right border of the sternum, because the commonest form of the disease results in immense hypertrophy and dilatation of the right chambers. (2) Palpable and sometimes visible pulsation over almost the whole of the cardiac area may often be detected for the same reason. (3) A loud, rough systolic murmur can generally be heard, loudest in the third or fourth interspace, close to the left of the sternum, and it is often easily heard in the back. These signs in a child who has a tendency to cyanosis are almost certainly due to cardiac malformation. Very loud bruits may mean but slight abnormality, whilst severe disease may be unaccompanied by even a slight murmur when the heart muscle is weak. (4) Dyspnœa is also fairly common, and may be either persistent or paroxysmal. The condition, however, may remain latent for many years, until exertion or some illness reveals its existence. The diagnosis is sometimes a matter of difficulty. Other symptoms arise as the disease progresses—thus, general cyanosis, reaching a very extreme degree ; coldness of the extremities ; syncope and epileptiform attacks ; a low temperature of the surface generally, although not of the interior of the body (Peacock) ; dropsy occasionally ; hæmorrhages from the lungs ; and symptoms of congestion of the other viscera. Dilatation of the conjunctival vessels is often observed, and clubbing of the toe and finger ends. Headache is often present, sometimes convulsions. Backwardness or precocity, and sometimes graver mental defects become evident as the child grows older.

Etiology.—Congenital disease of the heart arises under two conditions : (1) *Inflammatory affections* attacking the fœtal heart *in utero* may lead to stenosis of the orifices, almost invariably on the right side of the heart, which is in contradistinction to the left-sided lesions of extra-uterine life. (2) An *arrest of the closure* which normally takes place shortly after birth, of the ductus arteriosus, foramen ovale or the ventricular septum.[1] Three results follow : (1) Deficient oxygenation, and probably admixture of venous and arterial blood ; (2) the right ventricle takes an *equal* share with the left in the work of the heart, and consequently it hypertrophies and dilates ; and (3) the ductus arteriosus remains patent, to compensate for the insufficient delivery of blood into the aorta or pulmonary artery, as the case may be. It is only occasionally possible to suggest the precise nature of the lesion during life, but this, although of great interest, is not always of great moment.

Prognosis.—The condition may remain latent for many years, though few marked cases survive to adult life. The prognosis is serious in proportion to the degree of dyspnœa and cyanosis, pointing to deficient aëration of the blood, and in proportion to the other symptoms of " cardiac failure " (§ 55).

The *Treatment* is the same as that of Cardiac Dilatation (§ 50).

FALLACIES IN THE DIAGNOSIS OF SYSTOLIC MURMURS.—1. *Hæmic* murmurs (§ 402) are undoubtedly extremely common, and sometimes very difficult to distinguish from those of cardiac valvular disease.

2. A systolic murmur audible in the *aortic area,* and having all the characters of II. above, is due not so often to aortic stenosis as to (i.) roughening of the valve in old

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[1] Some observers maintain that the primary mischief is always the non-closure of the ductus arteriosus or foramen ovale or ventricular septum, narrowing of the aorta and pulmonary arteries being secondary.

people from *atheroma* or calcareous deposit ; (ii.) atheroma, with *dilatation* of the aorta, may also produce a systolic or a double bruit : then there is a ringing second sound in addition.

3. A systolic murmur heard best at the second left interspace is sometimes present in *mitral regurgitation* with a hypertrophied left auricle. It must not be mistaken for pulmonary stenosis.

4. The " *milk-spot* " murmur is due to a localised thickening of the visceral pericardium, appearing as a glistening white spot near the centre of the anterior surface of the heart. Usually it is unattended by symptoms, but it may be of importance clinically, for it is apt to be mistaken for valvular disease. The " milk-spot " murmur (based on twenty-three observations, verified by autopsy, at the Paddington Infirmary) is generally a prolonged rough bruit, systolic in time, though occasionally double ; it is *strictly localised* to a circle of 1 or 1¼ inches radius, whose centre is situated in the third left interspace, close to the sternum, which is also its position of maximum intensity. Another important feature is that *at one time it is very rough and loud*, and a day or so later it may have completely disappeared. These features, and the absence of the concomitant symptoms of cardiac valvular disease, or of chlorosis, enable us to differentiate the milk-spot murmur from other conditions. It was found more often in hypertrophied hearts than in those of normal size. It has been variously
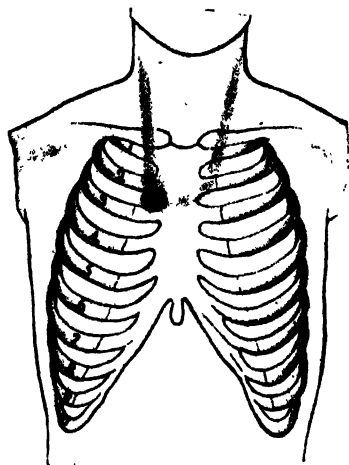
FIG. 17.—The systolic murmur of aortic stenosis. Depth of shading indicates intensity of murmur.
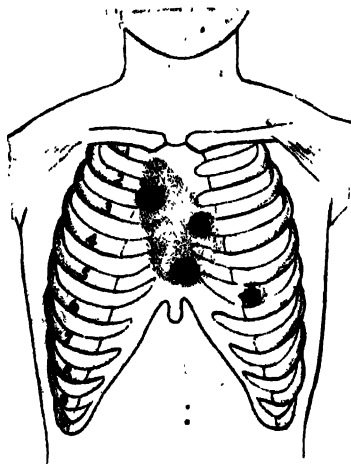
FIG. 18.—The diastolic murmur of aortic regurgitation. Depth of shading indicates intensity of murmur.

attributed to tight-lacing, the soldier's shoulder straps, and other less probable causes. The condition is more frequently met with in adult or advanced life. A history of pericarditis was obtainable in only one of the twenty-three cases.

5. A *congenital* murmur, usually systolic and localised to the base, has been known —in rare cases—to persist throughout life in some persons who have never experienced any other manifestation of cardiac disease, although they have lived to a good old age.

6. A systolic murmur heard shortly after the first sound may be heard at the apex at one stage of mitral stenosis (Mackenzie).

7. *Cardio-pulmonary or Cardio-respiratory* murmurs are also rare, and are probably produced by the expulsion of air from the adjacent lung tissue by the movements of the heart. They do not indicate any cardiac lesion, and the lung may also be healthy ; but they are sometimes associated with phthisis when the cavity is near the heart.

They are heard in various parts of the antero-lateral region of the chest. They have a blowing, whiffing, or "sipping" character, are usually systolic in rhythm, and in rare cases double, though the systolic element is always loudest. Often they are not loudest at the apex, and come rather between the two sounds than with the first sound. Sometimes they disappear when the patient alters his position or stands up. When he stops breathing, they may be weakened, abolished, or unaltered.

B. **Murmurs** heard in the **diastolic interval** may occupy either (a) the first half of that interval, replacing, accompanying, or following the second heart sound (*Diastolic* murmurs); or (b) they may occupy the second half of the interval, preceding and leading up to the first heart sound (*Presystolic* murmurs (see Fig. 19).

• Murmurs of the *first* kind are produced, in order of frequency, by aortic regurgitation, aneurysm, and pulmonary regurgitation. In Mitral Stenosis an early diastolic murmur is common. Murmurs of the *second* kind are mostly due to mitral stenosis, very rarely to tricuspid stenosis.

I. In **Aortic Regurgitation** the murmur is *diastolic* (Ventricular Diastolic).[1] (i.) The diastolic murmur produced at the aortic valve must be listened for : (a) over the lower part and to the left of the sternum. It may be audible as far as the apex and indeed over the whole heart. This murmur is usually to and fro in character and is found typically in those cases of aortic regurgitation in which the whole valve is more or less damaged. (b) Over the junction of the second right costal cartilage with the sternum. Here the murmur is harsh, loud, and just beneath the stethoscope. This is particularly associated with damage to the anterior cusp, the lesion most frequently liable to be connected with pains in the chest (angina). (c) Over the pulmonary base (third left costal junction). Here the murmur is soft, blowing but distant, never harsh in character. It is indicative of damage to the left posterior cusp, which is practically always, in the writer's experience, secondary to a mitral lesion. This murmur should be carefully listened for in any case of mitral stenosis, where the face is pale, where the left ventricle is large, where the pulse is medium to large in volume, or where the blood-pressure is high (J. S. G.). (ii.) Owing to the amount of dilatation and hypertrophy of the left ventricle, the apex is displaced downwards and outwards more than in any other form of valvular disease. (iii.) The pulse is the characteristic "water-hammer" (§ 73). The face is pale, the carotids visibly pulsate. Capillary pulsation is generally present, and is detected by drawing a line across the forehead, or by lightly pressing on the finger-nail or on the lips with a glass slide ; the alternate blush and pallor due to the pulsation in the capillaries is thus well brought out. So great may this be that a pulse is sometimes communicated to the veins on the dorsum of the hand. (iv.) In Aortic regurgitation the *blood pressure* is characteristic—the systolic pressure being high, the diastolic pressure low ; the pulse pressure is consequently high. Moreover, the systolic pressure in

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[1] *Diastolic* murmurs are sometimes spoken of as V.D. murmurs, being produced during the ventricular diastole. Similarly, *presystolic* murmurs are spoken of as A.S. murmurs, being produced during the auricular systole.

the leg is always higher than in the arm; in the " young " [1] rheumatic aortic, this difference is frequently as great as 100 m.m. In " young " rheumatic cases the high systolic pressure is due to the increased output and increased force of the enlarged left ventricle; in " old " cases (i.e. cases that start after middle life, when the heart has comparatively little power of hypertrophying) the raised pressure is largely due to the increased peripheral resistance. Hence in the " young " cases the high blood pressure is a measure of the left ventricular wall; in the " old " cases, it is largely an estimate of the peripheral damage. The increased pressure in the leg is usually due to vasomotor hypertonus; the difference between arm and leg pressure is usually taken as a measure of vasomotor reserve. A falling systolic pressure in aortic regurgitation usually means a failing myocardium; an approximating arm and leg pressure usually indicates approaching vasomotor collapse.

General Symptoms in aortic regurgitation: (i.) Pain about the chest, often of an anginoid character, may be complained of, or true angina may be present. (ii.) The pallor is greater in this than in aortic stenosis. Faintness, giddiness, frontal headache, and disturbed sleep are common. (iii.) Dropsy is rare, as death usually occurs before the mitral valve yields sufficiently to produce the necessary back pressure. Embolism sometimes occurs, though not so often as in mitral stenosis.

II. **Mitral Stenosis** is characterised by narrowing of the mitral orifice with obstruction to the free passage of blood from the left auricle to the left ventricle. Two primary varieties are described: (*a*) A congenital form, extremely rare, results from either malformation of the mitral ring or a fœtal endocarditis. (*b*) An acquired form, following (i.) a rheumatic infection, or (ii.) a slow, degenerative, sclerosing process, part of a generalised cardio-vascular degeneration.

Physical Signs.—The appearance of the mitral stenotic is often characteristic. In the rare *congenital* type the patient is small and undergrown (so that at the age of fourteen, for example, he or she appears little more than a child of seven or eight) with a red face, blue lips, cold blue hands with more or less marked clubbing. The *acquired* form, the ordinary rheumatic type, is most frequently met with in women. · The face is more or less pinched, there is a marked malar flush, whilst the tip of the nose, ears and extremities are cold and blue. Respirations are frequently marked, and the jugulars are often prominent. The *sclerosing* type is most common in men over forty. The patient has a typically thin face, pale or yellowish, the temporal arteries are tortuous and prominent and the jugulars engorged. The " mitral face " is typically a red face, but not infrequently Mitral Stenosis appears with a pale face. The pale Mitral Stenosis should always be regarded with suspicion as, generally speaking, it means one of the following: (*a*) an acute primary infection; (*b*) a secondary infection; (*c*) an associated aortic leak, or (*d*) some independent condition such as associated renal disease or anæmia.

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[1] *i.e.* those who contract rheumatic infection when young.

Physical Examination.—(1) The *pulse* in Mitral Stenosis is character-istic,. and gives a sure guide to the condition of the systemic circulation and left side of the heart. The *volume* is small (estimated by "lift" plus duration of wave), due to the diminished output. The *force* is small (estimated by impact against the finger), due to feeble contraction of the left ventricle. The tension is low (estimated by obliteration force), owing to diminished output and diminished force. The *rhythm* may be regular or irregular ; in the latter case the irregularity is due either to ectopic beats or to auricular fibrillation. The *rate* is usually rapid (round about 90), occasionally slow (40 or below) if heart-block supervenes. (2) The blood pressure is usually low, owing to diminished output and force of the left ventricle. It is particularly low and feeble after auricular fibrilla-tion has set in. In the sclerosing type the blood-pressure is high in pro-portion to the amount of peripheral damage. Should mitral stenosis become complicated by aortic regurgitation, the blood pressure tends to rise and the pulse volume and force increase.

(3) *Cardiac Signs.*—*Inspection :* Epigastric pulsation is often visible owing to the. dilatation and hypertrophy of the right ventricle ; the apex-beat itself can often be seen inside the nipple line, somewhat diffuse in character. *Palpation :* The apex-beat is slapping in character and well inside the nipple line. Typically, a presystolic thrill, and more rarely a diastolic, is felt at the apex. The presystolic thrill may be inter-mittent in character and only brought out by exercise, deep breathing, rest or change of position. An intermittent thrill occurs at the beginning and end of auricular compensation. In the former case the rhythm of the heart is regular ; in the latter usually irregular. The pulmonary valve closes so forcibly that it can usually be felt. *Percussion :* The cardiac dulness is slightly if at all increased. The right cardiac dulness measured from the mid-sternal line is increased. On the left, the cardiac dulness does not extend out to the nipple line unless there is some other associated condition such as mitral or aortic regurgitation, pericarditis, etc. *Auscultation :* The first sound at the apex is sharp, reduplicated or markedly accentuated and preceded by a crescendo murmur. This presystolic murmur, typical of mitral stenosis, is heard only over a limited area, and is not conducted outwards. In the later stages of the disease a diastolic murmur is audible in the region of the apex. Presystolic and diastolic murmurs frequently exist together (Fig. 19). The second sound at the apex is inaudible in a well-established mitral stenosis, while at the base the pulmonary second sound is markedly accentuated and reduplicated. In the sclerosing type of mitral stenosis the aortic second sound is frequently accentuated, while a rough systolic murmur due to roughening of the aortic ring is not infrequently heard.

General Symptoms.—The commonest symptoms [1] associated with mitral stenosis are : (1) Dyspnœa, at first only on exertion after meals, later after ordinary exertion (*e.g.*, stairs). It then becomes continuous

[1] Based on a critical examination of 884 cases.   J. Strickland Goodall.

and progressive, so that the patient is unable to lie down at nights (orthopnœa). Violent sudden dyspnœa occurs in pulmonary infarction. . (2) Palpitation is at first intermittent; occurring after exertion, and is simply of the nature of a physiological tachycardia. Later on it becomes more or less continuous, occurring independently of effort. It is then frequently associated with auricular fibrillation, flutter or paroxysmal extrasystoles. (3) Cough is a common symptom, induced by exercise or change of position. It may be associated with the spitting of blood and marked cyanosis. The underlying cause is generally congestion. A severe type of cough is sometimes met with in embolism or infarction. (4) Liver engorgement and ascites are common.

In order to understand the progressive variations of the physical signs and symptoms met with in Mitral Stenosis, it is necessary to say a few words about the anatomical changes which develop in this lesion. Broadly speaking, narrowing of the mitral orifice from any cause results in : (a) a tendency to dam up the blood in the left auricle, pulmonary and venous circulation generally, so that the venous pressure tends to rise ; (b) a reduction in the left ventricular input, so that the left ventricle is badly filled, badly stretched and inadequately stimulated to contract. The output
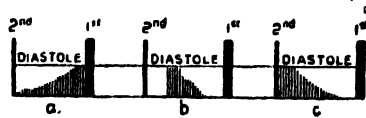
Fig. 19.—Three murmurs may be met with in MITRAL STENOSIS, which may occupy different parts of the diastolic interval, and may therefore be called the (a) LATE, (b) MID, and (c) EARLY DIASTOLIC MURMURS. The presystolic murmur is present in the early stage ; the early diastolic is a later event. The reduplication of the second sound has been omitted for the sake of clearness.

and force are thus reduced and the systemic blood-pressure falls. The damming of the blood in the pulmo-venous circulation is responsible for the physical signs met with in the chest, e.g., accentuated pulmonary second sound, basal crepitations, etc., as well as the epigastric pulsation, engorged jugulars, enlarged liver, etc., frequently met with. The reduced left ventricular input is responsible for the apparent small size of the left ventricle, the position and character of the apex beat, the small low tension pulse and low systemic blood pressure. The progressive changes which occur in the myocardium in Mitral Stenosis and its actual condition at any phase of the disease can easily be made out by a critical study of the electrocardiogram. The first result of Mitral Stenosis is increased auricular work and contraction and this is reflected in the electrocardiogram by increase in the amplitude of the P or auricular waves, which still however retain their normal form (Fig. 20, I). As the condition progresses, the two auricles become slightly divorced in their action so that the P waves become flattened and partly divided (Fig. 20, II and III). Usually by the time this has occurred a contracting mitral ring has definitely involved the Bundle of His, with impairment of its conductivity, as is shown by a definite increase in the PR interval (Fig. 20, III). Usually also, by now right ventricular " compensation " has occurred, so that the electrocardiogram shows a distinct right-sided preponderance (Fig. 20, III). The narrowed mitral orifice results in imperfect filling and therefore incomplete stretching of the left ventricle which consequently contracts poorly (hence the slapping character of the apex beat) and this is mirrored in the electrocardiogram by poorly marked T waves in Lead 2 (T²), the altitude of these being much below normal. The exaggerated, flattened and delayed P waves, the diminished T² waves and marked right-sided preponderance giving rise to a picture which is characteristic of Mitral Stenosis (Fig. 20, III, Leads i, ii and iii). The condition continues to progress, the muscle fails, the rhythm becomes irregular—the first irregularity noticed usually

being due to extrasystoles of either auricular or right ventricular origin (Fig. 20, IV).
This indicates myocardial hyper-irritability of either auricle or right ventricle, and
unless rapidly and efficiently treated is succeeded at an early date by a complete
irregularity due to auricular fibrillation which may or may not be immediately pre-
ceded by a state of flutter (Figs. 20, V and VI).   One would emphasise the fact that
extrasystoles occurring in a case of Mitral Stenosis are always to be looked upon as
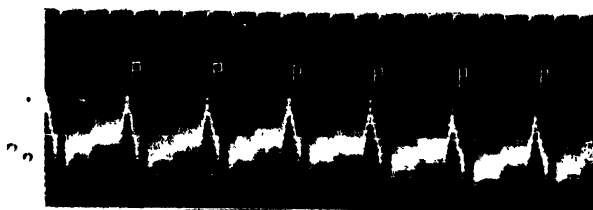
I.   Early Mitral Stenosis Large P (Auricular) Waves.
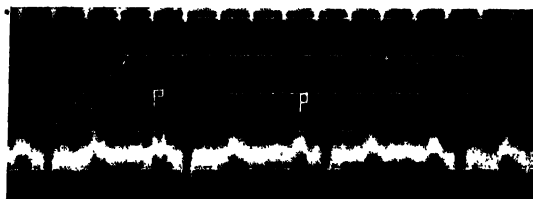
II.   Slightly later stage showing divided P Waves.

Ld. i

Ld. ii
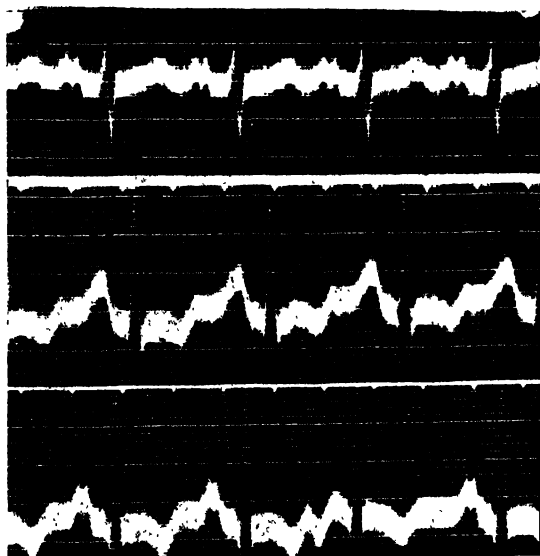
Ld. iii

III.   Shows all the typical features of a well-established Mitral Stenosis, viz. right-sided preponderance
(deep " S " waves lead I—tall " R " waves lead III).   Large delayed " P " waves.   Increased
PR interval and small inverted " T " waves (lead III).

FIG. 20.—MITRAL STENOSIS.

Fig. 20 (*continued*).—MITRAL STENOSIS.
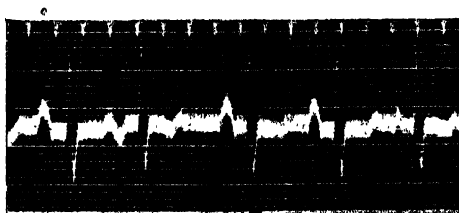
IV.   Shows commencing irregularity due to Auricular Extrasystoles (E.).
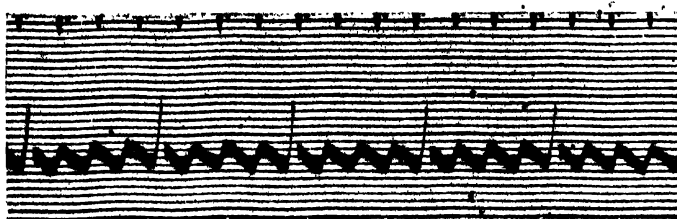
V.   Auricular Flutter.
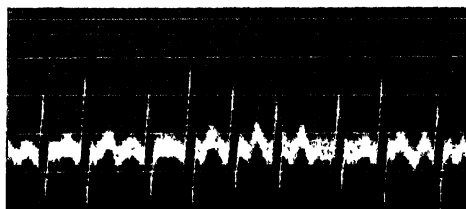
VI.   Auricular Fibrillation.

indications of impending trouble.   The fibrillation at first is of the coarse variety, but as time passes become finer and ultimately flat as the muscle gradually degenerates and its co-ordination and contractibility become more impaired.   Definite heart block is not uncommon.   (J. S. G.)

III.   In AORTIC ANEURYSM a *diastolic* murmur is sometimes heard if the aortic ring shares in the dilatation of the aorta.

IV.   TRICUSPID STENOSIS is a rare condition, but it is occasionally met with in young women, and is recognised by (i.) a presystolic murmur, heard loudest over the fifth right costal cartilage, close to the sternum.   (ii.) Dropsy is an early effect, but in other respects the consequences are the same as those of regurgitation through this orifice.

V.   REGURGITATION through the PULMONARY ARTERY is practically never met with, excepting either as an accompaniment of congenital malformation of the heart, or as part of a general valvular inflammation in acute ulcerative endocarditis.

FALLACIES IN THE DIAGNOSIS OF DIASTOLIC MURMURS.—1. A diastolic murmur due to *aortic regurgitation* may be heard at the *apex*.   It must not be mistaken for that of mitral stenosis.   In addition to the fact that the aortic murmur is heard louder at the base than at the apex, it has a uniform character, whereas a mitral diastolic murmur is of varying intensity, and the character of the pulse and other symptoms are different.

2. A *presystolic apical* murmur is occasionally heard with aortic regurgitation

(Austin Flint murmur). It is diagnosed from that due to mitral stenosis by its not being followed by an accentuated first sound, by the position of the cardiac impulse, and by the absence of the other signs of mitral stenosis.

3. *Mitral stenosis* is the most difficult form of valvular disease to detect in the late stages, when the characteristic murmur may be *altogether absent.* It may, then, be strongly suspected when there is—(i.) a loud, clear, sharp first sound at the apex, with marked accentuation of the pulmonic second sound ; or (ii.) hypertrophy of the right ventricle, chronic pulmonary catarrh, and hæmoptysis, especially if the second is reduplicated or absent at the apex.

* C. Double Murmurs may be produced by a combination· of any of the above systolic and diastolic murmurs.

.(*a*) Double murmurs most audible at the **base** (other than hæmic) :

I. COMBINED AORTIC OBSTRUCTION AND REGURGITATION is the most common condition, and causes a loud double see-saw murmur, heard best in the second right interspace.

II. ANEURYSM OF THE AORTA may be attended by a double murmur having the same characters as in disease of the aortic valves. This is heard loudest in the second right interspace, but it may also be heard at the back, to the left of the fourth dorsal vertebra.

III. A double murmur occasionally occurs in the DILATED AORTA of the aged, but with less marked features.

IV. A double murmur, loudest in the pulmonary area, usually indicates CONGENITAL HEART DISEASE.

(*b*) A double murmur most audible at the **apex** may be heard when both MITRAL REGURGITATION and STENOSIS are present. It consists of a systolic bruit followed by a long diastolic murmur almost filling up the diastole.

FALLACIES IN THE DIAGNOSIS OF DOUBLE MURMURS.—1. When a double murmur can be heard both at *the base and apex*, do not imagine that mitral regurgitation exists, as well as aortic disease. Remember that a systolic mitral and a systolic aortic may be alike in character, and that aortic murmurs can often be heard at the apex, as well as the base. To arrive at a conclusion is often very difficult, but one must rely on the position in which the murmur is loudest, and on the other features which distinguish mitral and aortic lesions.

2. When *a double aortic* murmur is present, the lesion may be regurgitation, or stenosis, or both together. A diagnosis is made by examining the pulse (§ 68), the time of the thrill, if one is present, and the position of the apex-beat. In regurgitation the apex is displaced farther downwards and outwards than in any other form of valve disease. In aortic stenosis the left ventricular wall is hypertrophied, with but little enlargement of the cavity, but as emphysema is so often associated with it, the apex may be hard to find.

3. Murmurs of *pericardial friction* may easily be mistaken for a double aortic murmur.

4. *Hæmic, cardio-pulmonary,* and *milk-spot* murmurs are occasionally double.

§ 55. GENERAL SYMPTOMS OF CARDIAC VALVULAR DISEASE.—The first effect of valvular disease is *hypertrophy* of the heart, as already mentioned, and so long as there is adequate compensatory hypertrophy there may be no concomitant symptoms at all.

But, sooner or later, in most cases hypertrophy gives way to *dilatation*, and then a series of characteristic symptoms ensue. Those special to each form of valvular lesion have been referred to in the preceding section. Certain *general symptoms are common to all forms of chronic valvular disease.*

1. *Breathlessness* on walking uphill, or even on very slight exertion, is a very constant feature. No serious enfeeblement of the heart wall or disturbance of its function can exist withowt this symptom ; and it cannot be too much insisted on that breathlessness is not only a symptom, but, in general terms, is a measure of the extent of the cardiac failure.

2. *Dropsy* occurs early in mitral, late in aortic, disease.

3. *Palpitation* is of less diagnostic import, for it may occur without any organic heart change, and is not always present with valvular disease.

4. *Pain* is by no means always present in cardiac dilatation, but few cases run their entire course without considerable præcordial discomfort. Pain is a fairly common feature of aortic disease, and sometimes amounts to angina.

5. *Insomnia*, in advanced cases, is frequently a very troublesome symptom. Sometimes the patient, when dropping off to sleep, suddenly starts with the terror of suffocation, and gasps for breath. *Headache* and *delirium* are also met with in advanced cardiac disease. The former is occasionally due to temporary high tension, but both are more often due either to pyrexia or to a toxic condition of the blood from failure of the emunctories. In either case free purgation is indicated.

6. *Embolism* may occur, having all the features described under Acute Endocarditis (§ 45). It is most frequent in mitral stenosis, and next in aortic disease.

The chief **Cause** of cardiac valvular disease in *youth* is acute endocarditis, which has a special tendency to attack the *mitral* valve, and in *advancing years* the chronic degenerative changes which attack the *aortic* orifice, and may gradually spread down upon the adjacent mitral cusp.

1. *Acute Endocarditis* of rheumatic origin is by far the most frequent cause, and a large majority of " heart cases " date their symptoms from an attack of that disease in youth or early adult life. Scarlatina and the other acute specific fevers, and all causes of acute endocarditis (§ 45), play their part, but the other specific fevers are infrequent relatively to acute rheumatism and scarlatina.

2. Chronic Endocarditis may come on insidiously, especially under the influence of *certain poisons*, chief among which are alcohol, syphilis, and gout, and especially if these be combined with hard labour. Under such circumstances the lesion usually affects the aortic orifice. But chronic endocarditis more often supervenes upon acute endocarditis—an attack of which may have been overlooked.

Endocarditis may start with an apparently trivial attack of subacute rheumatism, the child complaining of nothing but slight pains in the limbs, or in one joint, accompanied perhaps with a slight sore throat, not of sufficient gravity for him to be kept

in bed. Parents whose antecedents are rheumatic should be warned not to treat such symptoms lightly.

3. *Degenerative* changes (*e.g.*, atheroma) are the lesions chiefly met with after middle life. They affect especially the aortic orifice, either by injuring the valves or by causing dilatation of the aorta, which, extending to the situation of the valves, prevents them from meeting during diastole.

4. Any prolonged *high blood-pressure*—*e.g.*, that which accompanies arterial sclerosis —may lead to valvular strain, usually aortic. Persistent obstruction in the lungs (*e.g.*, chronic bronchitis), or in the general systemic circulation, may have the same effect as persistent high tension on the right or left side of the heart respectively.

5. Extensive or prolonged *muscular exertion* may, it is believed, lead to valvular mischief--at least, there is no other mode of explaining the fact that a large number of athletes have sclerosis of the aortic valves. In rare circumstances a sudden strain may lead to rupture of a valve.

6. *Congenital* conditions are referred to in § 54.

Prognosis of chronic valvular disease. Statistics are misleading if they are based only on hospital cases. Patients need to be followed from beginning to end as in private practice or infirmary work. It is quite certain that many patients have disease of the heart for years without knowing it. It is also certain that the first symptoms very often date from the patient knowing that he has cardiac disease, and unless there are special reasons to the contrary, a patient should never be informed of its presence.

Cardiac disease may terminate life in three ways : (i.) By sudden death - ·this may result either from syncope, or from rupture of the heart, or, as some say, from cardiac anæmia, due to non-filling of the coronary arteries ; (ii.) by the occurrence of complications, especially bronchitis, and other pulmonary affections ; or (iii.) by asphyxia, from dropsy of the pleura, often combined with congestion of the lungs.

The probable course and duration depend upon many considerations, but on nothing more than the condition of the *cardiac wall* (No. 3 below), and this should be the object of the most thorough investigation (see § 38).

1. The presence of certain *Cardiac Symptoms* is in itself an indication that the reserve power of the cardiac muscle is overdrawn—*e.g.*, palpitation, dyspnœa, increased by emotion or exertion, cardiac pain, syncopal and anginoid attacks. In actual practice the prognosis is good in proportion to *the amount of exercise a patient can take without producing breathlessness*.[1] Syncope and anginoid attacks usually indicate serious cardiac embarrassment. Palpitation and cardiac pain are less serious indications ; " Delirium cordis " and Cheyne-Stokes breathing are very grave.

2. The *Condition of the Pulse* is of considerable value in prognosis, but it has to be judged in connection with the valvular lesion. Irregularity is of varying significance (§§ 61 and 71).

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[1] Mackenzie states : " The simple test is to observe how the heart responds to effort. . . . Heart failure is first exhibited by a diminution of the work force of the heart, and this is shown by a restriction of the field of effort."

3. The *Condition of the heart muscle* and reaction to treatment (§ 56).

4. The presence of *signs of venous obstruction* as an indication of backward pressure—viz., pulmonary congestion, dropsy, lividity of the lips and fingers, enlargement of the liver and spleen, and albuminuria—is unfavourable. But the gravity is very different in mitral and aortic lesions respectively. In *mitral* cases a moderate degree of these symptoms indicates only moderate cardiac failure, and it by no means follows that the heart is beyond redemption. But if they occur in *aortic* disease they show that the final stage is reached, and that the patient will probably not live many months. When general venous congestion exists, the relative *amount of urine* passed day by day is a good measure of the strength of the heart and the improvement made—a fact which is not generally appreciated.

5. Concerning the *Nature of the Valvular Lesion* as bearing on the prognosis, some difference of opinion is expressed as to the relative importance of aortic and mitral lesions. My own experience is that a moderate degree of aortic stenosis is the most favourable form, and if well compensated may give rise to little or no inconvenience ; the patient generally dying of some intercurrent malady. Next in order comes mitral regurgitation, then mitral stenosis ; the most serious being aortic regurgitation, the valvular disease which most frequently ends in sudden death. Combined lesions of stenosis and regurgitation are naturally more serious than single ones, and the gravest of all valvular lesions is double aortic disease.

In *Aortic Regurgitation*, the *measure of the amount* of regurgitation, and therefore the prognosis, depends upon the clearness with which one can hear the aortic second sound (as distinct from the murmur) in the carotid arteries, and on the degree to which the pulse collapses. In *Mitral Regurgitation* a loud murmur *following* the first sound is more favourable than a weak murmur, or than one which replaces or accompanies the first sound. Apical murmurs due to atonic *dilatation* can generally be made to disappear under treatment.

In *Double Aortic Disease* it is important to note which is the louder, the first or the second of the two bruits. If the first be the louder, it indicates considerable compensating hypertrophy of the left ventricle, and the prognosis is more favourable; but if the second (the regurgitant) bruit be the louder, it probably indicates a weakened ventricle, which allows a large reflux of blood, and the prognosis is as grave as well can be.

6. The *Primary Cause* of the valvular mischief influences the prognosis to some extent. Injury and congenital mischief, both happily rare, are very serious. Rheumatism is grave in proportion to its tendency to recur. In general terms cases due to acute endocarditis in early life are much more favourable than those due to the degenerative changes (accompanied perhaps by an alcoholic or syphilitic taint) supervening during middle life.

7. *Age* is not a very important factor. Valvular lesions in childhood are more readily compensated, but at the same time advance more rapidly. *Mitral stenosis* coming on in childhood is much graver than when it supervenes in the adult, and

generally terminates fatally before the age of thirty. On the other hand, *aortic regurgitation* due to endocarditis in youth is compatible with a long and useful life ; but when coming on in middle or advanced life, it is generally due to degeneration and dilatation of the aortic orifice—a condition of far graver import.

8. The *Temperament, Habits,* and *Means* of the patient will naturally influence his future. Want of rest and sleep, the presence of worry and other causes of nerve strain, seriously affect the prognosis in all forms of cardiac disease. The prognosis is bad in the intemperate, and those who lead irregular lives. It is also unfavourable in the destitute, and in those who are compelled to work hard for their daily bread. Nevertheless, complete idleness is equally bad, and a patient should be encouraged to do as much as he is able without fatigue.

9. Finally, before hazarding a prognosis in any given case, the *Effects of Treatment* should always be watched, for it is sometimes truly wonderful how the skilful administration of digitalis, and the application of modern methods of treatment, will sometimes seem to snatch the patient from the very jaws of death. The existence of an organic murmur without change, and not requiring active treatment for two years, justifies a favourable prognosis.

§ **56.** The **Treatment** OF CHRONIC HEART DISEASE (including Myocardial Degeneration and Valvular Disease) may be considered under three heads : (a) When compensation is fully established ; (b) when compensation begins to fail ; (c) when compensation has broken down.

(a) When there is efficient compensation, no symptoms are present and no active treatment is needed, but much may be done to prolong the patient's life, and to avoid the supervention of cardiac failure. Subjects of chronic valvular disease should be enjoined to lead quiet, regular, and orderly lives. They should be warned particularly against the dangers of any *sudden*, unusual exertion, such as running to catch a train. With regard to exercise, it may be said, in general terms, that the patient himself is the best judge, provided always that he does not exert himself sufficiently to cause palpitation, severe dyspnœa, or præcordial pain. Some sports are more permissible than others ; thus cricket, tennis, and golf may usually be enjoyed, whilst football, racing, and rowing must generally be forbidden. Climbing, especially to high altitudes, must be disallowed. Alcohol, tobacco, and tea are all myocardial poisons if taken to excess, and should be used only in strict moderation. The skin should be kept active by the daily bath, and the bowels regular by means of purgatives if necessary. Whenever possible, a means of livelihood should be chosen in which the heart is subjected to but little strain. A sedentary occupation with moderate exercise in the intervals, is more suitable than one which entails earning a living literally by the sweat of the brow. Lifting or carrying heavy weights, climbing ladders, wielding heavy hammers, and physical labour in constrained positions, are liable to overtax the powers for compensation of the cardiac muscle. Meals should be regular, and heavy meals should be avoided. A short rest should be taken before and after every meal. The diet should be easily assimilable, and contain only a moderate amount of fluid. Anything requiring prolonged digestion disturbs the night's rest, so that it is sometimes a good rule to allow nothing solid after 5 p.m. for those who have

a tendency to indigestion or gastric disturbances. A small quantity of stimulant with meals may be called for, but should not be used unnecessarily, because of the reaction afterwards,·and of the tendency to excess, which exists in cardiac cases.

(*b*) When compensation is beginning to fail, the condition of the heart should be noted frequently ; rest, drugs, and exercises being prescribed in accordance with the variations in the circulation and the capability of response to treatment by the cardiac muscle.

Drugs.—In cardiac failure, especially in auricular fibrillation, when the pulse becomes feeble, rapid, and irregular, digitalis is *par excellence* the remedy. It is especially indicated in failure of the right heart and in mitral regurgitation, whether primary or secondary 'to aortic lesions. It is contra-indicated when there is full compensatory hypertrophy, and the pulse is fairly strong, regular, and slow, or, if vomiting is present. The work of Sir James Mackenzie has thrown much light upon the action and modes of administration of digitalis. It slows the heart, and improves contractility ; in overdoses it induces heart-block. It should be given in large doses (ℨ i. daily (4)) to get the muscle thoroughly under its influence ; the dose is then reduced to the minimum which experience and careful observation prove to be capable of maintaining the regularity and slowness of the pulse. It is especially useful in auricular fibrillation, except where there is pyrexia or fibroid degeneration, when the muscle does not respond well to the drug. It should be continued for a considerable time in smaller doses in the form of a tonic. Strophanthus and other cardiac drugs are less efficacious than digitalis. The action of digitalis and many other cardiac remedies is expedited by an occasional dose of calomel. Formulæ 54, 57, 59, 67, and 84 are useful. The action of digitalis is slow, and in very acute cases of heart failure recourse must be had to rapidly acting stimulants, such as brandy or ether, caffein citrate gr. ii–iij (0·13–0·19), or camphor gr. iii (0·19) in oil subcutaneously, digitalin $\frac{1}{100}$ grain (0·0006), strophanthin gr. $\frac{1}{150}$ (0·0004) may be given intravenously. Strychnine is useless as a cardiac stimulant. In *aortic valvular disease* and in the early stages of *mitral stenosis*, digitalis is not so valuable a drug ; but in the later stages of these affections, when compensation begins to fail, and especially when auricular fibrillation is present, digitalis gives relief. In aortic cases, where the blood-pressure is high, or where angina is present, the vaso-dilators are often of much use, such as nitroglycerine in the form of liquor trinitrini ℳ i. (0·06) t.i.d., erythrol-tetranitrate or sodium nitrite. Belladonna is sometimes useful if there is constriction of the vessels. Among the general tonics strychnine, nux vomica, iron, and arsenic are the most valuable in the order mentioned.[1]

[1] Cane-sugar in the form of glebe granulated sugar in doses of 1 to 5 ounces twice or three times daily is strongly recommended in failing myocardium from whatever cause, by Dr. Goulston (*Brit. Med. Journ.*, March 18, 1911), and Dr. Carter (*Brit. Med. Journ.*, November 25, 1911).

The various symptoms may be met by appropriate remedies.   For the *pulmonary congestion*, squills and stimulating expectorants are indicated. For *breathlessness*, spirits of ether or of chloroform and ammonia are useful.   Nitroglycerine is useful where breathlessness is associated with high tension, which may accompany cardiac hypertrophy ; and at the same time it cures the headache and sleeplessness due to the same cause.   Ether ♏xx. to xl. (1·3–2·6), or strych. sulph. gr. $\frac{1}{60}$ (0·001) hypodermically, are useful for the paroxysms of dyspnœa.   *Cough* is relieved by drinks of hot milk, and drugs such as codeia, small doses of opium, and chloroform or ether.   For *palpitation* alcohol is a most valuable cardiac stimulant, and relieves the breathlessness as well.   Unfortunately, patients soon find this out for themselves, and thus cardiac valvular disease is a not infrequent cause of chronic alcoholism, especially among women, who take it secretly, during the night, when the palpitation is most apt to come on.   The exact dose should therefore be carefully prescribed, and the quantity should always be moderate.   Other causes of palpitation which may be present should be treated (§ 24).   For *sleeplessness* opium or morphia hypodermically is most useful, and should be given without hesitation.   In children or in cases where the insomnia is not obstinate, other drugs may be employed, such as potassium bromide, sulphonal, trional, and paraldehyde.   I have not found small doses of chloral do harm, as some maintain.   The *hæmoptysis* of heart disease is best left alone, as it relieves the congestion.   The *gastric* symptoms may be relieved by acting on the congested liver with calomel, ½ to 1 grain (0·03–0·06) every night, with sodium sulphate and sodium bicarbonate (30 grains (2) in 2 ounces (64) of hot water) in the mornings.   Digitalis must be stopped if it causes sickness.   It may be necessary to give predigested food.   For the treatment of *syncopal attacks* and *pain, vide* §§ 26 and 25.   Formula 56 is useful.

Massage and Systematised Exercises.—At one time rest was regarded as imperative for all forms of cardiac disease.   But the advance of physiological knowledge has shown what an important part the skeletal muscles play in the circulation of the blood, by squeezing the fluids out of the soft-walled veins and lymphatics, while they cannot compress the lumen of the firm-walled arteries.   There are three varieties of this treatment, which are invaluable for different degrees of cardiac failure.   *First*, for the worst cases, *gentle massage*, combined perhaps with *passive movements*.   These are available where any kind of voluntary movement on the part of the patient is attended with breathlessness.   The great value of properly regulated massage in cardiac failure is well seen in the case from which the tracings in Fig. 21 were taken.   *Secondly*, slow *voluntary movements* of flexion and extension on the part of the patient while standing or sitting.   In the Nauheim system these voluntary movements are gently resisted by the operator—" resistance gymnastics."   These movements, combined with *baths* (see below), constitute the essence of the system.   *Thirdly*, Oertel's method, which consists of three parts : first, reducing

the amount of fluid taken to 31 ounces per diem (to include the amount contained in the solid food) and promoting perspiration; secondly, a diet largely consisting of proteids [1]; and ,thirdly, graduated exercise in the form of walking uphill, each day a little farther. Cases attended by plethora and obesity are the most suitable.

Saline and effervescent baths may be usefully added to the preceding. They act by relaxing the arterioles of the skin directly, and the arterioles of other parts reflexly. By these means blood is transferred from the venous to the arterial system, and its flow accelerated (F. 113).

(c) When compensation has broken down and marked cardiac failure is present, absolute rest is necessary. The patient is usually unable to lie down, but has to be propped up with pillows, and in severe cases sleep can be obtained only when the legs are hanging down. In severe failure of the right heart, as indicated by orthopnœa, lividity, distended jugular veins, the liver dulness extending well below the costal margin and the
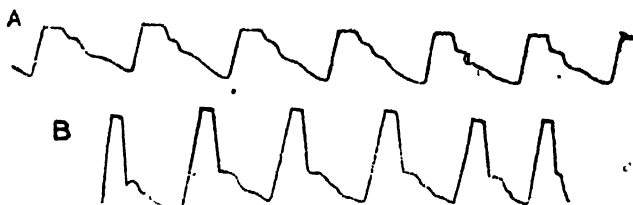
Fig. 21.—Tracings A and B show the efficiency of bleeding. A shows the flat top of high tension. B was taken immediately after 5 ounces of blood were removed, and shows the reduction thus effected, and also the senile character of virtual tension. The patient was about fifty-seven years of age, and suffered from cardiac valvular disease with recurrent high tension (with headache, etc.). The urine was always normal. Some years later he was brought in with apoplexy and died.

cardiac dulness extending far to the right, *venesection* is called for, and brings prompt relief. The abstraction of from 5 to 10 ounces of blood is usually sufficient; its efficacy is shown in Fig. 21, above. Three to six leeches may be applied to the right lower ribs in children,;in whom vene-section is more difficult to perform. The dropsy may require special treatment, such as draining the legs by Southey's tubes (§ 23) or multiple superficial incisions. Aspiration of a pleural effusion or ʿparacentesis abdominis may be necessary. Diaphoretics are of little use in cardiac dropsy. Cardiac tonics, such as digitalis and caffein, should be employed in conjunction with diuretics, calomel, and hydragogue cathartics, such as pulv. jalapæ co. and cream of tartar. The digitalis, squill, and calomel , pill is useful at this stage ; so also Formula 55. Diuretin and theocin-sodium acetate in 10 grain (0·6) doses are valuable drugs when dropsy is

[1] Oertel's dietary is as follows :—*Morning :* 6 ounces of coffee, 3 ounces of bread. *Noon :* 3 to 4 ounces of soup, 7 to 8 ounces of roast meat or poultry, salad or green vegetable, a little fish, 1 ounce of bread or farinaceous pudding, 3 to 6 ounces of fruit ; no liquid (excepting in hot weather, 6 ounces of light wine). *Afternoon :* 6 ounces of tea or coffee (1 ounce of bread occasionally). *Evening :* One or two lightly boiled · eggs, 1 ounce of bread, salad, fruit, sometimes a small piece of cheese, 6 to 8 ounces of light wine, with 4 to 5 ounces of water.

excessive. The diet should contain a minimum of salt. The readily diffusible stimulants such as spirits of nitrous ether, alcohol and sal volatile are of great value.
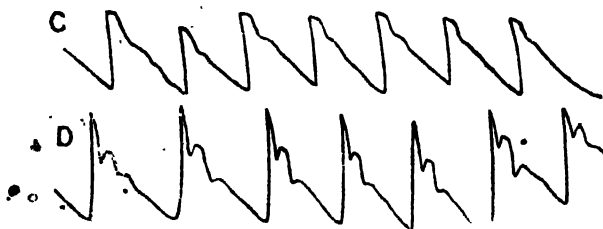

FIG. 22.—Pulse tracings C and D are taken by a Marey's sphygmograph (in which the momentum of the lever is greater and the excursion larger, than those taken by Dudgeon's). C (which shows simply high tension) was taken before, and D (which shows the reduction of tension) was taken directly after massage—massage and passive movements. The patient was a man aged sixty-five, under care for arterial sclerosis, and these tracings show the efficacy of massage in relieving the heart.

§ 57. E. We now turn to the consideration of those cardiac disorders the recognition of which depends upon **Alterations in the Rate or Rhythm of the Pulse.** In all cases it is essential to compare the radial pulse with the heart-sounds, and to observe the pulsation in the veins of the neck.

The Polygraph and the Electrocardiograph (§ 39) may be required to make an exact study of a case presenting pulse alterations; but it is often possible to make a correct diagnosis without their aid.

I. With an occasional PAUSE in the radial pulse .   .   .   .   } Premature beat (Extrasystole).

II. With RHYTHMIC alteration of rate DEPENDENT ON RESPIRATION .   .   .   .   .   . } Sinus Arrhythmia.

III. With INCREASED rate .   { Tachycardia, Paroxysmal and simple. / Auricular Flutter. / Auricular Fibrillation.

IV. With DISORDERLY RHYTHM . Auricular Fibrillation.

V. With DECREASED rate .   { Bradycardia :   Stokes-Adams' syndrome.

VI. COUPLING of the Pulse Beats   { Premature Beats. / Pulsus Alternans. / (Extreme Dicrotism.)

The various causes of altered rate and rhythm of the pulse, other than cardiac disease, are considered in § 69. Here we consider only the cardiac conditions to which attention may first be called, and in which the diagnosis may be largely made, by alterations in the pulse-rate and rhythm.

1. *There is an occasional pause in the radial pulse, during which the heart gives a short premature beat.* The condition is EXTRASYSTOLE.

§ 58. **Premature beats (Extrasystoles)** are due to hyper-irritability of the myocardium, causing early contraction in some part of the heart. The patient may or may not be conscious of the altered heart beat, and complains of palpitation, a catch in the breath, or uneasy sensations over the heart.

Broadly speaking, the *causes* of hyper-irritability which give rise to extrasystoles or premature beats are of (1) extrinsic, or extra-cardiac; or of (2) intrinsic, or cardiac origin. Pressure, *e.g.*, the stomach distended with gas, presses on the right ventricle and sets up a state of hyper-irritability, indicated by premature beats starting in either the right ventricle or auricle. Such beats disappear at once on relief of the distension. Again, in the eighth month of pregnancy the heart is almost always irregular from this cause.

The commonest intrinsic causes of hyper-irritability are : (1) Toxæmia ; (2) exhaustion ; (3) inflammation ; (4) degeneration.

(1) The connection between extrasystoles and *toxæmia* is appreciated when one considers such conditions as malaria, typhoid, influenza, excessive tobacco, etc., in which the irregular action of the heart is almost invariably due to extrasystoles. Generally speaking, all parts of the heart are involved, so that the irregularity is considerable, sometimes the auricles, sometimes the right and at other times the left ventricle showing premature beats. During the war, cases of disordered action of the heart (D. A. H.) of this type were frequently seen. The influence of tobacco in producing cardiac irregularities is well known and was a recognised method during the war of evading military service.

(2) The relationship between extrasystoles and *fatigue* is especially interesting. The premature contractions may be associated either with a general fatigue and only occur after actual physical effort, or they may be associated with either general or local myocardial exhaustion. Extrasystoles which appear or increase only after physical effort, indicate myocardial fatigue or damage. Any irregularity of the heart which develops after or upon exercise, whether due to extrasystoles, auricular fibrillation, auricular flutter or any other cause, must always be looked upon as dangerous and indicative of some definite myocardial disability. There are many interesting points in connection with more or less localised cardiac fatigue (cp. mitral stenosis, p. 86).

(3) *Inflammation.*—In acute myocarditis extrasystoles are common. In the chronic forms of myocarditis, such as that met with in syphilis, extrasystoles frequently occur independently of any endocardial or pericardial involvement. Persistent extrasystoles increased by exercise always indicate myocardial damage. If auricular, they often precede fibrillation ; if ventricular, they are often associated with a gumma or a diffused myocarditis. The association between extrasystoles and inflammation is often well seen in the course of an attack of rheumatic fever. The patient, perhaps, has well-marked signs of endocarditis or pericarditis, but as far as one can tell the myocardium has escaped damage. The heart then becomes irregular and owing to the development of extrasystoles, one can say that the inflammation has involved the myocardium.

(4) In *myocardial degeneration* extrasystoles most commonly occur after fifty years of age, when the cardiovascular system is degenerated ; they are frequent antecedents to such conditions as auricular fibrillation or heart block, associated with generalised myocardial change.

The beat of the heart always starts at the most irritable point, normally the sino-

auricular node. Should any other point of the heart become more irritable it initiates the contraction ; such a beat is said to be ectopic in origin and inasmuch as the contraction occurs earlier in the cardiac cycle than the normal beat, it is sometimes called *premature.* Roughly speaking, ectopic beats may arise either in the auricular, nodal or junctional tissue (*i.e.* the tissue between auricles and ventricles) or in the right or left ventricles. Ectopic auricular beats may or may not be followed by ventricular contraction. Should the ectopic beat start in the nodal tissue, it travels upwards towards the auricles and downwards towards the ventricles, so that the auricles and ventricles contract more or less simultaneously.

• The *prognosis* has been briefly mentioned under each cause. Here one may sum up thus : As auricular extrasystoles are almost always due to a pathological condition of the myocardium, they are often persistent. They may give rise to paroxysmal tachycardia ; this in turn is likely to produce fatigue and failure, which is specially serious if associated with aortic lesions or extensive ventricular myocardial damage. A point to be emphasised, therefore, is that hyper-irritability is a definite stage or phase in certain physiological or pathological processes ; and the extra-systole, which is an expression of myocardial hyper-irritability, must be regarded as a forcible demonstration that this stage has been reached. Secondly, extrasystoles, whether of intrinsic or extrinsic origin, are important because they frequently produce sudden increased intravascular strain, with disorganisation and embarrassment of the circulation. Ectopic beats are important according to (1) the underlying condition on which they depend, and (2) the amount of intravascular circulatory disturbance they produce (J. S. G.).

II. *The patient is* YOUNG, *and presents a* REGULARLY RECURRING *alteration of the pulse-rate, usually dependent upon* RESPIRATION. *The condition is* SINUS ARRHYTHMIA.

Sinus Arrhythmia is a condition in which the discharge of impulses from the sino-auricular node occurs at slightly varying intervals, producing a rhythmic irregularity of the heart.

• *Symptoms.*—The pulse-rate varies with respiration, but there is no great difference between the strength of any two successive beats. The regular waxing and waning of the pulse is often accentuated when the patient breathes deeply. Auscultation reveals no alteration in the heart sounds. Jugular vein tracings are normal. The patient may have attacks of giddiness or syncope or may have no symptoms at all.

Causes.—The condition is common in the young and during convalescence from diseases in which the heart-rate has been rapid. It is of vagus origin, and it is met with in nervous individuals especially, in whom there is exaggeration of the normal inspiratory increase and expiratory slowing of pulse-rate.

Prognosis.—The condition is of no importance ; it ceases when the pulse-rate quickens from any cause, *e.g.*, after exercise. When found after fevers, it is a good sign, inasmuch as it suggests the absence of extensive damage to the heart wall. No treatment is indicated.

§ **59.** III. *The Cardiac conditions in which an Increased Rate forms*

the most striking feature, are : PAROXYSMAL TACHYCARDIA, AURICULAR FLUTTER, AURICULAR FIBRILLATION.

In the majority of cases of regular TACHYCARDIA the increase is physiological in character. In two conditions, paroxysmal tachycardia and auricular flutter, the heart beat starts from a new focus.

These two forms of tachycardia may be differentiated by the following features :—In the physiological type the pulse-rate is (i.) affected by posture, falling 10 to 30 beats when the patient passes from a standing to a recumbent position ; (ii.) the pulse-rate increases with exercise, and is affected by emotion, meals, fever, and sleep ; (iii.) the onset and termination are gradual ; (iv.) electrocardiograms are normal ; (v.) jugular tracings show no exaggeration of the force of the auricle. The causes of this form of tachycardia are dealt with in § 69.

The pulse-rate is REGULAR, 160 *or more ; the rate is unaffected by alteration of posture. The condition is* PAROXYSMAL TACHYCARDIA OR AURICULAR FLUTTER.

Paroxysmal Tachycardia is a term which is now reserved for cases of rapid action of the heart presenting the following characters : (1) the onset of the tachycardia is abrupt ; (2) the duration varies from a few seconds to several days ; (3) the relief is sudden, and the pulse returns to its normal rate in the course of a few beats, which are often irregular in force and rhythm. During the paroxysm violent jugular pulsation is visible.

The *symptoms* complained of by the patient depend upon the duration of the paroxysm. Many of the short paroxysms, lasting a few hours, provoke no subjective sensations. When the attack is prolonged over several days, grave cardiac embarrassment with dilatation, cyanosis, œdema of lungs, and great engorgement and enlargement of the liver occur. Occasionally there is great distress and discomfort. The general disturbance of the patient, the rapidity of the pulse, and the severity of the abdominal pain dependent upon the engorgement of the liver may be so extreme as to simulate an acute abdominal condition calling for surgical interference. Cases are on record of exploratory laparotomy having been performed owing to such an error of diagnosis. The rapidity of the disappearance of the abdominal symptoms on cessation of the tachycardia is a very striking feature. The immediate *prognosis* depends chiefly upon the presence or absence of dilatation of the heart. The most severe symptoms may disappear in less than an hour if the heart wall remains intact.

Etiology.—The condition is due to sudden rhythmic activity of some definite focus of primitive tissue (usually in the auricle), which for a time overcomes and replaces the normal activity of the sino-auricular node. It is most common in young adults, but may occur in early childhood or old age. The attacks may be excited by exertion, emotion, flatulence, or change of posture. The disturbance is usually, but not always, a consequence of infective processes—the most common being rheumatism, scarlet fever and syphilis. Often no valvular lesion is present ; if valvular murmurs are present they become unrecognisable during the paroxysm. Post-mortem examination has shown fibrosis, pallor, friability of the heart-muscle and usually some coronary disease.

The *diagnosis* from tachycardia of purely nervous origin depends upon : (i.) the abrupt onset and relief ; (ii.) the presence of violent jugular pulsation ; (iii.) occasional presence of a few premature beats in the intervals between the paroxysms. Many attacks of so-called " Paroxysmal Tachycardia " are really paroxysms of Auricular Fibrillation or Auricular Flutter (see below).

Treatment.—The brief paroxysms which produce no subjective symptoms call for no treatment. For the prolonged attacks the patients often discover for themselves

some simple procedure which cuts them short, such as holding the breath, compressing the abdomen, or the assumption of some special posture. If these are inadequate, pressure may be made upon the vagus in the neck. Morphia may be called for and should embarrassment of the right heart become extreme, venesection and the removal of ℥ x.-xx. of blood may give relief.

§ **60. Auricular Flutter.**[1]—This name has been given to an uncommon condition in which " the normal beats of the auricle are submerged by contractions of this chamber in response to a series of new, rhythmic, and pathological impulses varying in rate from 200–350 per minute " (Lewis). The distinction from Paroxysmal Tachycardia is arbitrary as regards rate, and is drawn at 200 beats per minute. Flutter differs, however, from Paroxysmal Tachycardia in that it is almost invariably associated with some degree of heart-block (§ 62) ; the auricle may, for instance, be beating at the rate of 300 per minute, while the ventricle responds with only 150 beats per minute. In other cases a higher grade of heart-block (4–1) may be present, and the pulse-rate be about 75 and regular. The rate of the auricle is absolutely regular ; that of the ventricle may be regular or irregular, depending upon the constancy of the degree of heart-block present. It will be readily seen what difficulty there may be in diagnosing this serious heart condition.

Diagnosis.—For the certain recognition of the disorder polygraphic or electrocardiographic records are necessary, but its existence may be suspected when (i.) the pulse-rate is 130–160, regular, and maintained for long periods, especially if associated with syncopal attacks ; (ii.) the ventricular rate shows no quickening after exercise or slowing on lying down. This constancy of the rate on alteration of posture is an important diagnostic feature.

Causes.—The condition is usually unassociated with much cardiac enlargement or evidence of valvular disease, but there are evidences of cardiac failure, such as shortness of breath. The patients are often past middle life, and are subjects of arterio-sclerosis.

Prognosis.—The duration of the condition varies. It may occur only shortly before death ; more frequently it persists for long periods, even for years. This long duration is a further diagnostic point of great importance. There is a danger of the ventricle suddenly assuming a rate equal to that of the auricle—a condition which may lead to syncopal attacks which may be fatal.

Treatment.—Digitalis in 20 minim doses (1·2) three times a day will often effect a cure ; it acts by producing auricular fibrillation (see below). If the drug is then withdrawn, fibrillation may suddenly cease and the heart resume its normal rhythm. Even if digitalis fails to produce fibrillation, it may be relied upon to reduce the ventricular rate.•

[1] Flutter has been placed under this heading because it is usually associated with a regular ventricular pulse. When varying grades of heart block are present this regularity in rate disappears, but in contrast to the pulse of auricular fibrillation regularity in force and volume persists.

IV. *The pulse is irregular, with* COMPLETELY IRREGULAR *rhythm, and beats at the rate of* 100 *to* 160 ; *the condition is probably* AURICULAR FIBRIL-LATION.

§ 61. **Auricular Fibrillation** is recognizable by (i.) Complete irregularity of the pulse ; (2) the difference between the apex and the pulse rates ; (3) increase in the irregularity by exercise ; (4) absence of the " A " wave on the venous curve or absence of the " P " wave on the electrocardio-gram. (5) Auricular fibrillation is usually associated with a low blood pressure, due to the small output, but in cases where the peripheral resistance is raised (*e.g.*, arterial sclerosis, renal disease) the blood-pressure may be high. Furthermore, auricular fibrillation is almost invariably associated with (6) increase in the cardiac dulness. It is the commonest irregularity met with in the out-patient departments of the hospitals in men over fifty. (7) Auricular fibrillation is often paroxysmal in character, the attacks lasting from a few minutes to hours, days or even weeks. Paroxysmal cases usually ultimately become chronic.

Etiology.—The auricle is composed of a number of intimately con-nected muscle fibres and a normal systole of the auricle consists of a system-atic and co-ordinate contraction of the auricle from above downwards, *i.e.*, from the sino-auricular to the auriculo-ventricular nodes. The muscle fibres are extremely intimately connected, so much so that some people regard them as forming a syncytium ; it is thus comparatively easy for the stimulus or contraction to travel from fibre to fibre. In auricular fibrillation this co-ordinate contraction no longer occurs, but individual bundles of fibres, or even individual fibres, contract inco-ordinately, so that the whole auricular wall appears to be trembling or quivering. This inco-ordination may arise in various ways. Thus in mitral stenosis, enormous distension of the auricles leads to mechanical separation of the fibres. In acute inflammatory conditions of the auricle, inflammatory products in the intercellular tissue interfere with conduction. In toxic or degenerative conditions of the muscle, extension of the normal stimulus may be interfered with ; there results irregularity in the filling and stimu-lating of the ventricle. Briefly, therefore, we may say that auricular fibrillation occurs : (1) secondary to valvular disease, especially mitral stenosis ; (2) in degenerative conditions of the myocardium, *e.g.*, cardio-vascular sclerosis ; (3) in toxic conditions, *e.g.*, Graves' disease ; (4) in the course of acute inflammatory conditions, *e.g.*, acute carditis ; (5) as a result of syphilis.

The *prognosis* depends chiefly upon (1) whether the underlying cause is removable or not. In cardio-vascular sclerosis the cause is not removable but progressive ; the prognosis is consequently bad. In Graves' disease, provided the disease is only in the toxic and not in the degenerative stage, removal of part of the thyroid will remove the hyperthyroidism and the auricular fibrillation will cease. In syphilis the cause is often removable. In mitral stenosis the immediately exciting cause of the

fibrillation is a rise of pressure in the auricles.   If this is promptly relieved (*e.g.*, by bleeding) the fibrillation will often stop, but will return because the primary cause (obstructed outlet) cannot be removed.   (2) The condition of the ventricular muscle.   If the auricular fibrillation is due to a more or less local condition of the auricle, the outlook is comparatively good.   But if the cause of the fibrillation (*e.g.*, cardio-sclerosis) has also affected the muscle, the prognosis is bad.   (3) The extent to which the ventricle is overstimulated by the erratically acting auricle.   Obviously the greater the ventricular rate and the larger the number of ineffective beats, the greater the over-work of the ventricle and the worse the outlook.   (4). Whether or not the fibrillation can be controlled by any means short of removing the actual cause.   In some cases, *e.g.*, mitral stenosis, it may not be possible to stop the fibrillation, but it may be possible to control it by treatment so that the ventricular rate is slow and the number of ineffective beats few.   In such cases the prognosis is good, the fibrillation making very little difference indeed to the conditions of life, etc.

Treatment.—From the preceding, it is obvious that the treatment of auricular fibrillation lies in attempting to remove the underlying cause. In some cases this is comparatively easy, in others it may be impossible. In hyper-thyroidism it can be stopped by removing part of the overactive thyroid.   In syphilis inunctions of mercury, iodide of potassium, or other anti-syphilitic remedies will often result in the cessation of the condition.   In mitral stenosis, on the other hand, the removal of the cause is, short of surgical procedure, impossible, but lowering of the intra-auricular pressure by bleeding, leeching, purging, together with the administration of large doses of digitalis will frequently stop the incoordinate contraction.   Quinidine, moreover, will occasionally stop cases of fibrillation.   If it is impossible to stop the actual fibrillation, it is usually possible to control its effect on the ventricle.   A convenient way to do this is as follows : take the rate of the apex and the rate at the wrist—the difference between these two is an estimate of the number of inefficient beats which exhaust the heart.   Next, give the patient large doses of digitalis, *e.g.*, 20 to 30 drops of the tincture once or twice a day until the apex rate falls to round about 50, or until the patient gets signs of digitalis poisoning (stomach ache, diarrhœa, vomiting or coupling of the beats).   Again compare it with the rate of the wrist pulse, and stop the digitalis for a few days until the rate begins to go up.   Compare the apex and the pulse rates repeatedly to ascertain the rate at which apex and pulse rates most nearly coincide.   The number thus obtained is the most effective working rate of the heart, and enough digitalis should be administered to keep the heart rate constant at this figure.   The patient must, of course, be kept in bed while the above is being carried out, and the observations should be repeated from time to time with a view to seeing if it is possible to reduce further the amount of the drug or the ventricular rate (J. S. G.).

V. *Conditions of the heart which are associated with a* SLOW PULSE.

§ 62. Slow pulse—between 40 or 50 or below—occurs in three· more or less common conditions. ·

(1) The heart of the well-trained athlete ; (2) Debilitating and exhausting diseases ; (3) Conditions of complete or partial heart block.

In the bradycardia of the athlete, the subject usually looks and seems physically very fit ; on exercise he shows no symptoms of distress ; and his pulse rate, instead of climbing as the exercise is increased, suddenly doubles. ·

(2) In the bradycardia of exhaustion, the response to effort is along physiological lines although out of all proportion to the effort. •. · ·

(3) In complete or partial **heart block** the rate is unaffected by exercise and signs of cardio-vascular degeneration are frequently present. Periods of unconsciousness (Stokes-Adams' fits) frequently occur.

Etiology.—Conductivity is specialised in the Bundle system described in the introduction to this chapter. Any part of this conducting tract may be damaged, a whole series of conditions arising therefrom (Fig. 23). Sometimes the stimulus appears to be blocked above the sino-auricular node, when the whole heart is silent and misses a beat —a condition spoken of as sino-auricular heart block. Again if the substance of the auricular wall is damaged as, for example, in an interstitial myocarditis, there is a delay in the passage of the stimulus between sino-auricular and auriculo-ventricular nodes—intra-auricular heart block. If the Bundle of His is damaged, then either partial or complete auriculo-ventricular block occurs. Furthermore, either the right or left main branch may be damaged, right or left bundle block resulting. Right Bundle block not infrequently occurs in aortic regurgitation, in right heart strain during anginal attacks, in chronic lung affections and in certain cases immediately prior to death. Finally, the terminations of the Bundle in the ventricular wall may be destroyed, when the condition is spoken of as terminal or arborisation block ; two varieties of this occur. All these conditions can be easily made out by means of the electrocardiograph.

The varieties clearly depicted in Fig. 23 are here summarised :—

1 *Supra-auricular* (Sino-auricular).
2. Auricular.
3. *Auriculo-ventricular :*
    (a) Temporary { Complete.
                       Incomplete.
    (b) Permanent { Complete.
                        Incomplete.
4. *Ventricular :*
    (a) Main Branch Block :
        (1) Right } Temporary or permanent.
        (2) Left }
    (b) Terminal or Arborisation.

The conditions of the Bundle which can produce heart block fall under three groups : (1) infective processes, (2) degenerative processes, and (3) the influence of drugs. The infective processes are numerous : rheumatism is the chief ; diphtheria, scarlet fever and syphilis are all common. Degenerative processes include fibrosis, the result of old rheumatic infiltrative processes, interference with nutrition through disease of the coronary vessels, and tertiary syphilitic lesions of the heart muscle itself. Digitalis has a pronounced action in lowering conductivity of the Bundle, an effect which is usefully employed in the treatment of auricular fibrillation.

In the milder forms the *prognosis* and the *treatment* are those of the associated

disease. Digitalis is usually contra-indicated, owing to its action in lowering conductivity, but in severe cases, where signs of gross heart failure are also present, and rest in bed proves insufficient, digitalis may be serviceable in virtue of its beneficial action upon the ventricular muscle. In cases in which the possibility of syphilitic changes can be entertained active anti-syphilitic remedies should be employed.

Complete Heart Block implies total interruption of impulse between auricle and ventricle. When this occurs the ventricle initiates its own rhythm. This is regular, and at a rate which varies from 24–36 (usually 28–30) in different patients, but remains remarkably constant in any individual case. Complete heart block is not infrequently associated with syncopal attacks—STOKES-ADAMS' DISEASE, first described by R. Adams in 1827. The patients are usually advanced in years, complain of dyspnœa, and have marked bradycardia, the pulse-rate ranging from 20 to 40. Any mental excitement is liable to bring on an attack. The breathing becomes stertorous, the face cyanosed ; there is dilatation of both pupils, rigidity of the body, accompanied by clonic movements of the limbs ; the pulse occasionally ceases for a few seconds, the jaw drops, and for forty to eighty seconds the patient is to all appear-

Sino-auricular Block.

S.V.C.

C.S.

S.A.

I.V.C.

Auricular Block.

Auriculo-ventricular Block.

T

Q

R　T′　l

Right Main Branch Block.

Left Main Branch Block.

S

Arborisation Block.
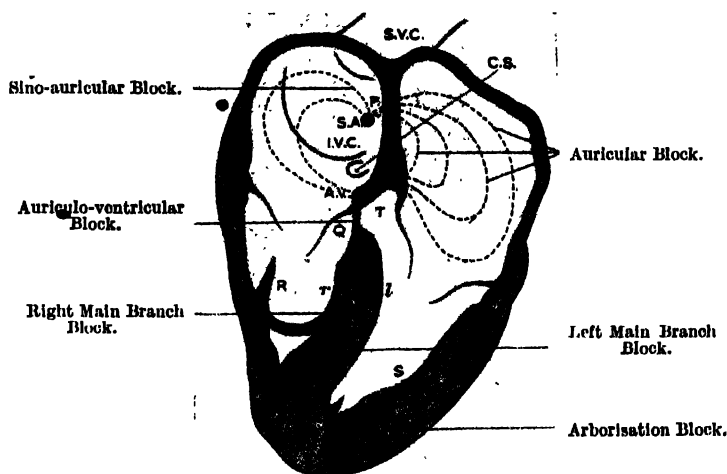
FIG. 23.—Diagram illustrating the positions of the lesions in the different varieties of Heart Block. (J.S.G.)

ance dead. No pulse is felt in either wrist, and on auscultation the cardiac sounds are inaudible· Then a feeble sound is heard, followed by a stronger, and a second later the pulse begins beating at about 30 per minute (one can feel the artery fill), the cyanosis lessens, the pupils contract, and consciousness returns. Many such fits may occur in succession, from six to ten in a single night. There is no albuminuria.

Treatment.—The attacks may be prevented by the administration of adrenalin which in all probability acts directly on the ventricle viâ the sympathetic. The underlying cause must, however, be treated ; for this purpose potassium iodide is often very useful.

§ **63.** *The Heart-beats occur in couples, with a pause after every alternate beat.*

This condition may be apparent or real, and may be due to :

(1) Regularly recurring PREMATURE BEATS ; a common cause of which is DIGITALIS.

(2) PULSUS ALTERNANS.

(3) Extreme DICROTISM produces an appearance of coupling, but may be distinguished from that due to premature contractions by the fact that the apparent second beat occurs synchronously with the closure of the aortic valves, and is unaccompanied by a systolic heart sound. It occurs only with very low blood-pressure (§ 73).

§ **64. Pulsus Alternans** is a condition in which every second beat of the heart is smaller and feebler than its predecessor. This sign is an evidence of exhaustion of contract-ility : it may be constant, or only appear occasionally after premature beats. It is distinguished from a coupled pulse due to regularly recurring premature contraction by the fact that the diastolic periods are constant throughout, there being no compen-satory pause after the small beat. The condition is of very grave significance when it occurs with a slowly acting heart. With a quick pulse-rate it need not be regarded with such grave apprehension, but its appearance is always a warning sign of cardiac exhaustion.

CHAPTER IV

ANEURYSM OF THE AORTA AND OTHER INTRATHORACIC TUMOURS

Anatomy.—The mediastinum is the irregular space in the chest which lies between the two pleural sacs. For descriptive purposes it is divided into four parts—viz., the *middle mediastinum*, which is occupied by the heart and pericardial sac ; the *anterior*, which is the space in front ; the *posterior*, the space behind ; and the *superior*, the space above the pericardial sac. The most important structures contained in these spaces are : The thymus or its remains ; the arch of the aorta with its branches (innominate, left subclavian, and carotids) ; the superior and inferior venæ cavæ, with the innominate and azygos veins ; the pulmonary vessels, the trachea and bronchi ; the vagus, recurrent laryngeal, phrenic, and splanchnic nerves ; the cardiac and pulmonary plexuses ; the roots of the lungs ; the œsophagus, thoracic duct, lymphatic glands and vessels, and loose cellular tissue (Fig. 9). The lymphatic glands are important on account of the occurrence of lympho-sarcoma and other glandular enlargements which may form mediastinal tumours.

If, on percussing over the sternum,[1] or just beside it, the præcordial dulness is found to be **increased irregularly upwards**—the morbid condition may be Pericardial Effusion, Enlargement of the Left Auricle, Retraction of the Lung, an Abdominal Swelling pushing up the Heart as a Whole, or Aortic Aneurysm or some other Mediastinal Tumour. The two last named are generally to be distinguished sooner or later by the presence of pressure symptoms (p. 112). If possible, a skiagram should be taken.

If, on auscultation over the **abnormal·dulness** *near the base of the heart, there is a* reinforced or ringing second heart sound—*perhaps a systolic or diastolic murmur—the disease is probably* Aneurysm of the Aorta.

§ 65. Intrathoracic Aneurysm.—Aneurysm of the aorta is undoubtedly the commonest of intrathoracic tumours. In regard to the anatomy of this serious and important malady, the student should study Fig. 9 (p. 49).

The arch of the aorta is the favourite seat for aneurysmal dilatation. Its shape and the fact that it is subject to continuous strain make it surprising that the malady is not even more frequent. Any part of it may be affected—the ascending, transverse, or descending part of the

[1] Remember, in percussing over the sternum, the note elicited is of a much higher pitch than that just beside the sternum.

arch. The dilatation may assume either a fusiform or saccular shape, the former being the more frequent. A fusiform dilatation arises as a rule in the first part of the aorta, and may lead to stretching of the valves and aortic incompetence. The fusiform aneurysm gives rise to practically no physical signs, and the ensuing description refers, unless otherwise stated, to saccular aneurysm. It may make its way in various directions, and it is extraordinary how bones, cartilages, and other hard structures may become eroded and absorbed under its pressure. One of the earliest results of aneurysm near the root of the aorta is *cardiac hypertrophy*, but this may not occur at all when it involves other parts. According to its position, aneurysm of the aorta may be either very easy or very difficult to detect. If it involves the first part of the aorta, near the *front* of the chest, it is soon revealed by definite *physical signs*. If the second or third parts of the arch are involved, and the tumour extends backwards, there may be no physical signs, and even the *pressure symptoms* may be obscure. Thus the clinical manifestations belong to two categories— physical signs and pressure symptoms ; and we have two varieties of aneurysm : (*a*) The *aneurysm of physical signs*, when the FIRST HALF of the arch is involved ; (*b*) The *aneurysm of symptoms* (that is, pressure symptoms), when the SECOND HALF of the arch is involved.

The *Symptoms Common* to aortic aneurysm in all positions will be considered first, because these are the symptoms which will probably first attract our notice. Then we will turn to certain others *special* to the first, second, and third parts of the arch respectively.

Symptoms COMMON TO ALL POSITIONS :

1. Dyspnœa is often one of the earliest complaints which the patient makes. When it is due to pressure on the trachea, as in aneurysm affecting the transverse portion of the arch, it is persistent and stridulous in character. When it is due to pressure on the anterior pulmonary plexus, as in aneurysm of the first part of the arch, it is often paroxysmal.

2. Cough is generally present and has a characteristic brassy sound (gander cough). Pressure upon the recurrent laryngeal nerve is common, with consequent paralysis of the left vocal cord, and there may be hoarseness or even aphonia from the same cause. *Paralysis of the left vocal cord* in the absence of central nerve lesions, practically always means aortic aneurysm. Laryngoscopic examination should be a matter of routine in all suspicious cases, because abductor paralysis occurs before complete paralysis, and the former may be unattended by any alteration of voice.

3. Pain in the chest is another common symptom. It may occur in attacks of an anginoid character, shooting down one or both arms, usually the left, especially in aneurysm of the first part of the arch. The pain may be neuralgic when there is pressure on nerves ; or it may be of a dull boring character when due to erosion of bone, such as occurs in connection with aneurysm of the descending arch.[1] Short of definite anginoid

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[1] A case is mentioned in the footnote to § 25 in which this was almost the only symptom.   G

attacks of this kind, patients with aortic aneurysm are liable to feelings of suffocation, constriction, or " spasm " in the chest, and nameless dreads come over them from time to time without cause. Such attacks may in many cases be brought on by bending the head backwards, or by any movement which stretches the neck. I have known patients with dilated and rigid aorta suffer from the same symptoms.

4. A reinforcement of the aortic second sound is the most constant of the auscultatory signs of aortic aneurysm. It is sometimes spoken of as a " ringing " second sound.

5. The diastolic shock or thud is an equally important sign. It is felt by the hand or the stethoscope, and is synchronous with the second sound.

6. Inequality of the radial pulses is a fairly frequent symptom. It is present whenever the aneurysm is so placed as to cause a difference in the arterial pressure in the great vessels which spring from the aorta. The typical aneurysmal pulse occurs in the vessel just beyond the sac, and its characteristic is a decrease of the pulse wave, the blood tending to flow in one continuous stream.

7. Inequality of the pupils occurs from inequality of carotid blood-pressure and corresponding inequality of blood-pressure in the vessels of the iris. In the early stages the irritation of the sympathetic nerve causes dilatation of the pupil on the same side. Later on there is paralysis, with contraction of the pupil, accompanied sometimes by vascular dilatation and unilateral sweating of the face and neck.

8. The heart may be displaced when the aneurysm is large, usually to the left. It is sometimes hypertrophied.

(a) Symptoms peculiar to aneurysm of the **ascending** or **first part of the arch.** Aneurysm of this part of the arch is usually easy of detection, and in marked cases the *Physical Signs* are unmistakable. (i.) On auscultation an accentuated second sound is usually to be heard ; and in a large number of cases, where the dilatation involves the valvular orifice, a diastolic murmur is also heard. Over the site of the aneurysm a systolic murmur is always present, and this is frequently present also at the aortic area. Thus, a double murmur at the aortic area is found in many cases. (ii.) Any percussion dulness present is continuous with that of the heart. It usually extends to the right of the sternum, but this depends upon whether the aneurysm makes its way forwards or not. The left heart gradually hypertrophies. (iii.) On palpation, the diastolic shock is very characteristic. Sometimes there is a thrill felt also in the suprasternal notch. (iv.) When the aneurysm is so large as to form a tumour, the swelling expands laterally with each systole of the heart. The accompanying erosion of the sternum may be very painful. (v.) The right bronchus may be pressed upon, leading to diminished or absent respiratory murmur (R. M.) of the right lung. In severe cases there may be pressure on the superior vena cava, with œdema of the neck and arms. (vi.) The dyspnœa is paroxysmal ; and the right recurrent laryngeal nerve may be involved, with right laryngeal paralysis.

(*b*) The symptoms of aneurysm of the second or **transverse part of the arch** may be equally easy to detect when it makes its way forwards. But when the posterior part is affected it may present considerable difficulty in diagnosis, especially from other intrathoracic tumours. (i.) The dyspnœa may be either paroxysmal or continuous, with inspiratory stridor, owing to the pressure upon the trachea. (ii.) Pressure upon the left bronchus may lead to diminished breath sounds in the left lung, partial collapse or bronchiectasis, and symptoms (2) and (5) above are specially marked in aneurysm of the transverse arch. (iii.) Tracheal tugging is-a very characteristic sign of aneurysm in this situation. Standing behind the patient, hold the cricoid between the finger and thumb, and press gently upwards, the patient sitting in a chair erect with the chin up (see Fig. 24). In this way the pulsation is transmitted by the trachea to the hand. (iv.) The physical signs—which are in this situation less marked, or may be absent—consist of a thrill felt on palpating the suprasternal

FIG. 24.—Tracheal tugging showing position of hands in order to elicit this symptom.

notch ; dulness on percussion over the manubrium, continuous with that of the heart, and extending from the middle line to the left of the sternum ; and auscultatory signs are described above—(*a*) (i.).

(*c*) The symptoms of aneurysm affecting the **descending aorta** may be very obscure. (i.) Intense pain in the back is the most common symptom, and there may be no other for a long time (*case* in footnote, § 25). The pain may pass to the side, following the course of an intercostal nerve. (ii.) Other pressure symptoms, such as dysphagia, from pressure upon the œsophagus ; wasting, from pressure upon the thoracic duct ; disease of the left lung, from pressure upon its bronchi ; and any of the other symptoms mentioned on p. 110. (iii.) If the swelling enlarges, physical signs on auscultation and percussion may become apparent in the left (occasionally the right) scapular region ; and in advanced cases there may even be a pulsating swelling without the knowledge of the patient. Osler found that in some cases there is absence of pulsation in the femoral arteries.

Etiology.—(1) Aortic aneurysm is far more frequent in men than in

women, especially in those in the prime of life—namely, between the ages of thirty-five and fifty. (2) It is especially frequent among soldiers and those who do laborious work. . This liability has been attributed to the wearing of belts and the like, but it is probably due to the fact that these classes are subjected to sudden and severe muscular exertion and heart-strain at certain times. It also occurs among blacksmiths for the same reason.

(3) Both syphilis and alcohol are potent agencies in the production of arterial degeneration. Alcohol acts probably in two ways—partly by predisposing to degeneration of the aortic walls, and partly by over-stimulating the heart from-time to time.

(4) Some cases of aneurysm date from a period of over-exertion, exposure, and destitution, or from an injury as an exciting cause.

Diagnosis.—The diagnosis of a deep-seated aneurysm is sometimes as difficult in the early stages as it is easy when the aneurysm is situated superficially. The diagnosis from *cardiac valvular disease* and other causes of cardiac hypertrophy (§ 49) is made by the occurrence of the pressure symptoms. Many of the local signs of a saccular aneurysm may be produced by a *dilated and rigid aorta*, but here the pressure symptoms are wanting. The *throbbing aorta* of aortic regurgitation is apt to be mistaken for aortic aneurysm, and it is sometimes impossible to differentiate these conditions. The throbbing aorta in Graves' disease and severe cases of anæmia may also give rise to difficulty. *Mediastinal growths*, on the other hand, may have the same pressure symptoms as aneurysm. Pressure upon the veins is more commonly seen with growth than with aneurysm, and may only be diagnosed by the absence of the physical signs referable to the heart. There is no murmur on auscultation over the dull region, the area of dulness is usually not so limited or defined, there is usually no expansile pulsation over the tumour, and there are signs of collateral circulation. Finally, the course of mediastinal tumours rarely lasts longer than eighteen months. Radiography is very valuable in the diagnosis of the presence and nature of intrathoracic tumours.

· *Prognosis.*—By treatment much can be done to prolong life, and the patient may live a good many years if his occupation does not necessitate much exertion. Death may occur in four ways—from rupture, exhaustion, cardiac failure, or complications. The rupture usually leads to a sudden and copious hæmorrhage, which terminates life ; but sometimes there is a slight leakage, which may recur at intervals of a few days. With aneurysm of the *ascending* aorta rupture usually takes place into the pericardium, pulmonary artery, or superior vena cava ; with aneurysm of the *transverse* arch, into the trachea (a very frequent situation) or into the bronchi ; and, when the *descending* aorta is involved, the blood usually finds its way into the pleura or œsophagus. The process may be so gradual that there is no sudden onset of symptoms, such as dyspnœa, or cyanosis, or bleeding, and death may not take place for some time. But generally, it is copious and sudden, death speedily ensuing. The severity of any

case is measured to some extent by the amount of dyspnœa present and the rapidity of the evolution of symptoms. Other consequences or complications are due, for the most part, to the effects of pressure--such as collapse or a low form of pneumonia of the lung, hydrothorax, and œdema of the head and neck.

Treatment.—The indications are three in number : (*a*) To lower the blood-pressure ; (*b*) to slow and steady the heart ; and (*c*) to increase the blood coagulability in the hope that laminated clot will form in the sac. Absolute rest in bed must be enjoined. This alone may accomplish very considerable relief, and there is no doubt that some of the extraordinary results claimed for certain remedies have been due to rest. Much can be accomplished by diet. It should be of the smallest quantity consistent with life. Tufnell's dietary [1] is based on this fact, and in it only 8 ounces of fluid and 10 ounces of solid are allowed per diem. It must be persevered in for three to six months. The good derived from this dietary mainly depends on the reduction of fluid. Drugs should be employed to steady the heart and reduce the blood-pressure (Pulse, § 72). There is, however, one remedy which is undoubtedly capable of materially relieving the symptoms of thoracic aneurysm- -viz., iodide of potassium in large and gradually increasing doses, commencing with 20 grains (1·2), three or four times a day.

The occasional administration of calcium chloride in doses of gr. xx. (1·2) t.d.s. for three days, in view of its known power to increase coagulation, is worthy of trial. The digestive organs often need attention. For the pain, morphia, atropine, or belladonna, internally or in the form of a plaster, are used ; if of anginoid character, nitroglycerine. Even if the dyspnœa is very urgent, tracheotomy is not called for unless the condition is due to bilateral laryngeal paralysis. If there be an external swelling, some elastic support is needed. Calomel is valuable for high blood-pressure ; aconite for palpitation. For venous distension or severe , dyspnœa venesection may be performed. Surgical measures have been adopted from time to time in the treatment of superficial aneurysms, but they are not free from danger. Of such we may mention acupuncture, galvano-puncture, and the injection of coagulating fluids—such as perchloride of iron (a dangerous procedure). Distal ligature of one of the great vessels sometimes leads to improvement, especially if it is involved in the aneurysm.

Other Mediastinal Tumours

§ 66. The **Symptoms of Mediastinal Tumour** belong to three categories—namely, (*a*) the signs of displacement of organs ; (*b*) the physical signs of tumour ; (*c*) the symptoms of pressure. There are also (*d*) certain symptoms special to the different kinds of tumour.

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[1] The solids may consist of well-cooked meat or fish and biscuit, and for the fluid 10 ounces of milk are permitted per day. From 12 ounces to 18 ounces solid may be permitted, but the fluid must not exceed 16 ounces. It must be combined with absolute rest, and drugs are better avoided.

(a) The **displacement** of organs is sometimes the first intimation we receive. The liver is rarely displaced, but the lungs and heart are often pushed to one side, and the apex-beat may be found in the axilla.

(b) The physical **signs of tumour** appear sooner or later on the anterior or posterior aspects of the chest, and consist of : (1) Dulness on percussion, corresponding to the position of the tumour; (2) auscultatory signs, which differ somewhat with the position and nature of the tumour. If it be solid, the breath sounds will be tubular and perhaps differ on the two sides, and there may be an increased conduction of the heart sounds. If it contain fluid (such as aneurysm or, more rarely, hydatid) there will be a diminished respiratory murmur, and in the case of aneurysm a characteristic murmur (§ 65). (3) Ausculto-percussion will aid in defining the boundaries of the tumour. (4) Radiography is used for defining the nature and position of mediastinal growths.

(c) The symptoms of mediastinal tumour which are due to **pressure** on the various structures around are as follows :

(1) Dyspnœa always appears sooner or later, and may be of a type peculiar to mediastinal tumours when there is pressure upon the trachea; it has a stridulous character, which resembles tubular breathing heard without the aid of the stethoscope. The breathlessness is often paroxysmal or asthmatic when there is pressure upon the heart and cardiac plexuses ; or it may be of a Cheyne-Stokes nature. But the character of the dyspnœa depends upon whether it is the heart, the great vessels, the bronchi, or the nervous apparatus of the heart, lungs, or larynx, which is pressed upon by the growth of the tumour.

(2) Cough, sometimes of a laryngeal brassy character, is also present, and it is accompanied by expectoration if, as is usual, there is also bronchitis or congestion of the lungs. There may be laryngeal paralysis from pressure upon the recurrent branch of the vagus, and hoarseness, or even aphonia, may result.

(3) Cardiac and circulatory symptoms, such as palpitation, cyanosis, or a difference in the pulses of the two sides in the neck or radial arteries. There may be signs of collateral circulation, with enlarged superficial epigastric and mammary veins.

(4) Dysphagia, from pressure on the gullet, is present chiefly with posterior mediastinal growths.

(5) Inequality of the pupils may appear, owing to pressure on the sympathetic. Usually the pupil on the affected side is contracted from paralysis of the sympathetic, but it may be dilated during the stage of irritation.

(6) Pleuritic effusion occurs if there be pressure on the thoracic veins or if there be growth in the pleura.

(7) The inferior vena cava is rarely compressed, but lividity or œdema of the head, neck, and arms may occur from pressure on the superior vena cava.

(8) In suspected tumour of the superior mediastinum, it is well to remember that when the head is thrown back, the veins of the neck become distended, owing to the increased thoracic pressure producing venous obstruction. Dyspnœa is marked, and the sternum may bulge forward.

(9) Pain down the arms and in the back occurs when there is pressure on the spinal nerve trunk.

(d) **Causes.**—There are certain symptoms which are **special to the nature and situation of the tumour.** There are five clinical groups of tumours, in addition to aortic aneurysm.

I. MALIGNANT TUMOURS, which may be primary or secondary. If, in addition to the above physical signs, the expectoration present a constant prune-juice character, and if on paracentesis a bloody fluid is drawn off from the pleura, the presumption is strongly in favour of malignant tumour. The fluid may contain cells recognisable as malignant. Out of 520 cases of mediastinal tumour, Hare [1] found 134 were cancerous.

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[1] Hare (*Mediastinal Tumours*, Philadelphia, 1889) found out of 520 cases, 134 were cancer, 98 sarcoma, 21 lymphoma, 7 fibroma, 11 dermoid, 8 hydatid, 115 suppurative mediastinitis.

Cancer of the mediastinum is the commonest mediastinal tumour, because it is usually secondary to cancer of the lung or œsophagus. In the latter case it is situated in the posterior mediastinum. Primary cancer, as of a bronchus, is rare, and tends to affect secondarily the anterior mediastinal glands. *Sarcoma*, especially lympho-sarcoma, may start in the mediastinal glands as a primary growth, or originate from the pleura and from the thymus remains. Primary sarcoma is most frequent in the anterior mediastinum. If secondary in origin (as when the abdominal viscera are the seat of the primary tumour), it occupies chiefly the posterior mediastinum. In primary mediastinal sarcoma enlargement of the glands in the neck and elsewhere may occur.

II. INNOCENT MEDIASTINAL TUMOURS, though more rare than the foregoing, are sometimes found, *e.g.*, fibroma, dermoid cyst, hydatid. Lipoma, gumma, and enchondroma, the latter growing from the sternum, are also occasionally met with.

III. ENLARGEMENT OF THE MEDIASTINAL GLANDS.—With these there is often a dulness posteriorly in the upper half of the interscapular space, but occasionally there is dulness over the sternum. Paroxysms of coughing, "croupy" or like whooping-cough, may be present, together with stridulous breathing from pressure upon the trachea. The *causes* of enlarged bronchial glands are:

(*a*) As described above, *malignant disease* of the glands is the most common cause of enlargement.

(*b*) *Tubercle*, which is generally secondary to tubercle of the lungs. It is more common in children than in adults. The condition may be suspected when con-current disease of the lungs is present, and symptoms such as the above arise. If the glands suppurate, sweatings and intermittent temperature become more pronounced than when the lung only is diseased. An abscess may form and open into a bronchus (compare IV. below).

(*c*) *Lymphadenoma (Hodgkin's disease)* may start in the mediastinal glands, and is then difficult to diagnose from lymphosarcoma. See also § 438.

(*d*) *Bronchitis* and the *pneumonia* which complicates measles, influenza, and whoop-ing-cough, are often attended by enlargement of the bronchial glands, which may occasionally be recognised behind the sternum in children.

(*e*) *Whooping-cough*, without bronchitis or other disease of the lungs, may give rise to swelling of the bronchial glands, although the condition may be hard to make out. Some observers consider that it is the pressure of these glands which causes the paroxysms of whooping-cough.

IV. SUPPURATIVE MEDIASTINITIS (abscess of mediastinum) is a rare condition which may affect the anterior or posterior mediastinum, or both, but more often the anterior. (i.) The most prominent symptom is pain, in the site of the inflammation, or passing down the nerves pressed upon. (ii.) Dulness, with œdema and redness, may be present over the upper part of the sternum if the disease be in the anterior region, or over the dorsal spines if in the posterior mediastinum. Pulsation communicated from the aorta may be present, and lead to a diagnosis of aneurysm, but the pulsation is not expansile, and fluctuation may be felt. (iii.) Pyrexia is present, usually of a hectic type, with the rigors, sweats and weakness which attend all deep-seated inflam-mations. (iv.) The presence of leucocytosis is an important diagnostic feature. The causes of the acute form of mediastinitis are trauma, erysipelas, and the eruptive fevers. The chronic form is usually due to tuberculous disease. It may rupture in various directions.

V. ENLARGEMENT OF THE THYMUS.—A certain degree of enlargement is normal to childhood, and may cause dulness over the manubrium. It begins to decrease after the second year of life, and should have disappeared by adult life. In lymphatism (§ 32) the thymus may be found enlarged even in adult life. An enlarged thymus is also frequently found in Graves' disease, and rarely in Addison's disease, myxœdema, myasthenia and rickets. Inflammation, œdema, and tubercle may affect the gland. Tumours may occur—cysts, sarcoma, rarely epithelioma, lymphoma and lymph-adenoma.

Prognosis.—In all cases of intrathoracic tumour which are large enough to produce symptoms the prognosis is unfavourable. Moreover, all of these conditions entail much suffering to the patient. Malignant tumours are fatal in six to twelve months, depending upon the site and progress of the growth. Innocent tumours may last for a long time. Syphilitic, tuberculous, and simple inflammatory glandular enlargements may recover under treatment, but even in these no confident prognosis of recovery can be given in any case. Suppurative mediastinitis may open externally, and run a course of a few days or weeks only; other cases are chronic, and last for years, or lead to pulmonary gangrene and other serious complications when the pus burrows into adjoining organs. An enlarged thymus may lead to sudden death from pressure upon the trachea.

Treatment in intrathoracic tumour is almost wholly palliative. For aneurysm, see § 65. Abscesses, hydatids, or growths connected with the sternum may be dealt with by the surgeon in some cases.

CHAPTER V

THE PULSE AND ARTERIES

§ **67. The Meaning of " The Pulse."**—By the term " pulse " is understood the expansile sensation communicated to the finger by the alteration in the shape of the artery, due to the momentary increase of blood-pressure which takes place during the systole of the heart, and which is transmitted to the periphery *in the form of a wave.* It has been shown that there is no dilatation of the artery ; the increased output of blood for the moment raises the blood-pressure, and alters the shape of the channel from an oval to a circle. The examination of the pulse is of extreme importance, not because it has a set of diseases of its own, but because it affords us so many valuable practical hints about the diseases of other organs, and about the general condition of the patient.

For the production of the pulse three factors are requisite ; (i.) The contractions of the ventricle, which determine the frequency and rhythm of the pulse, and to a large extent its force ; (ii.) the elasticity of the large vessels ; (iii.) the peripheral resistance found in the arterioles and capillaries. These three factors must always be considered in studying the pulse.

§ **68. Clinical Investigation.**—It is preferable not to examine the pulse until the preliminary excitement occasioned by the doctor's visit has subsided ; and in all accurate records the pulse should be noted under similar conditions as regards the posture of the patient, time of day, relation to meals, &c. The radial pulse is the one usually selected for examination, since it is easily accessible and lies against a bone. But the pulse can be observed in other situations—*e.g.*, the temporal, dorsalis pedis, or popliteal arteries. Three fingers should be placed along the course of the artery, the index finger next the heart, and allowance should. be made for much adipose tissue. The different means of eliciting the several features will be dealt with below. When feeling the pulse, its special features may often be brought out more fully by holding up both wrists with the fingers on the pulses. Only experience and comparison between all types of pulse can give to the physician the necessary aptitude for observation and correct inference. A complete observation of the pulse should comprise six features, the first four being the most important.

1. *Rate and Rhythm (i.e.,* regularity).—The rate of the pulse per minute is easily calculated by the watch, and in making this observation it should be remembered that a physiological acceleration occurs after any exertion, excitement, or after a meal, or may even be caused by

nervousness on the visit of the doctor. The pulse is faster in the evening than in the morning, and it is faster by about eight beats per minute in an upright than in a recumbent posture.[1] If the pulse be irregular, the type of the irregularity (vide infra) must be noted.

2. The Force or strength of the pulse is the measure of the systolic or maximum blood pressure. It can be estimated by its resistance to digital compression—the finger next the heart presses the vessel until the wave is no longer appreciable to the other fingers. By the amount of pressure required to obliterate the wave, the force with which the blood is propelled from the heart can be estimated. • A " full bounding pulse " is one which has strong pulsations, but it is not necessarily one of high blood-pressure. Indeed, a full bounding pulse may occur in fevers where the blood-pressure is generally low.

3. The Character of Each Beat is observed by noting (i.) whether the pulse wave rises suddenly or gradually ; (ii.) the duration of the beat, whether long or short ; and (iii.) whether the decline is abrupt or gradual. It is important to note the presence or absence of dicrotism, which is a marked feature in low blood-pressure (§ 73).

4. The state of the minimum Blood-Pressure, or diastolic pressure, is estimated by the degree of fulness of the artery between the pulsations. Normally the vessel is hardly felt between the beats if the wall is healthy. The vessel should be rolled transversely under the fingers, and in thin subjects and in many cases in which the arterial tension is high, it stands out like a cord between the beats. , This in the majority of cases is due to changes in the arterial wall which accompany a high pressure.

5. The Size of the Artery and the State of its Walls are considered later. It is important to note these features, because an artery of small size may give the impression of a weak pulse. The thickness of the wall must also be noted, because a thick-walled artery may give the impression of high arterial tension.

6. It should be part of the routine to examine the pulse of both Radial Arteries, as by this procedure we may detect the existence of unsuspected disease, such as aneurysm or other intrathoracic tumour. Abnormalities such as a more or less superficial position of the radial on one side or the other exist more frequently than is supposed.

The pulse is considered in the sections dealing with the examination of the patient with heart disease § 36. Here will be briefly mentioned the chief alterations of the pulse and their causes. I. Rapidity ; II. Infrequency ; III. Irregularity ; IV. Changes in the Blood-Pressure. The treatment belongs to the causal conditions.

The SPHYGMOGRAPH is an instrument employed to obtain a record on paper of the characters of the pulse. The first one used was that of Marey. In this instrument a

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[1] The pulse is faster in the female than in the male, and it varies considerably at different ages, thus : In the fœtus and new-born infant its average rate is 140 per minute ; under 1 year, 120 ; under 3 years, 100 ; from 7 to 14, 90 ; from 14 to 21, 80 ; from 21 to 65, 70 ; in old age, 80 per minute.

pad placed over the pulse is connected with the short arm of a lever ; the long arm, which magnifies the pulse wave, is sharpened to a point, and makes a tracing on smoked paper.

The handiest instrument, however, is that of Dudgeon. This is a little instrument which is strapped on to the wrist. Some years ago Weiss made for me a modification of the latter which can be used without a strap—an appendage which I regard as unnecessary, since the instrument can more readily, with a little practice, be steadied and adjusted by the hand of the operator. The manipulation of any of these instruments is easily acquired by experience. The chief precautions are : (i.) To place the pad *exactly* over the artery, and it is of great assistance if the course of the vessel has been previously marked by an aniline pencil ; (ii.) the *amount of pressure used*, and the adjustment of the instrument, should be such as to obtain the most graphic record.

The SPHYGMOGRAM or sphygmographic tracing is very useful as a graphic record of the pulse, and to show the progress of the case from day to day ; but it does not tell us as much as the educated finger, and its readings can never be quite accurate because the exact amount of pressure exercised by the pad upon the artery cannot be
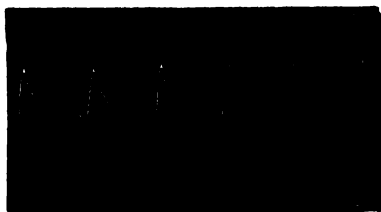
FIG. 25.
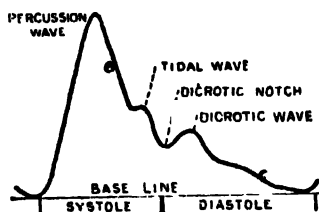
FIG 26.

FIG. 25.—NORMAL PULSE TRACING, taken with the author's modification of Dudgeon's Sphygmo-graph. Rate 68 ; pressure about 2 ounces. FIG. 26.—NORMAL PULSE TRACING (Fig. 25) magnified, with the names of the principal parts. The dicrotic (or aortic) notch indicates the closure of the sigmoid valves, and therefore the termination of the ventricular systole and the commencement of the ventricular diastole. The diastolic line is that part of the tracing from the dicrotic notch to the next percussion wave.

known. Figs. 25 and 26 represent a normal pulse tracing, the principal named parts of which it consists being indicated in the latter. (1) The *percussion wave* is abrupt and vertical in proportion to the force of the ventricular contraction and inversely proportional to the peripheral resistance. (2) The *tidal wave* is prominent in proportion to the amount of peripheral resistance and the force of the ventricular contraction —*i.e.*, when the arterial pressure is *high*. When the peripheral resistance is very low there may be no tidal wave. (3) The *dicrotic wave* is pronounced and the *aortic notch* more marked in proportion as the peripheral resistance and the heart force (*i.e.*, the arterial pressure) are *low*.

Many instruments have been devised for the MEASUREMENT OF THE BLOOD-PRESSURE. It is advisable to take two readings at the first attempt with a nervous patient, or indeed with any patient, and to regard the second as the more reliable. For practical purposes there are only two instruments which need be considered—the aneroid and the mercurial manometer. The aneroid type is portable and compact, but requires more frequent adjusting in order to ensure accuracy. The Tycos make is one of the best known of this variety. The mercurial manometer is the more reliable, and of these the Riva-Rocci sphygmomanometer is the type on which all are modelled. The column of mercury should be open to direct atmospheric pressure and so constructed that the mercury can be readily renewed and cleaned when necessary. The armlet should be at least five inches wide. The leather cuff is the more accurate, though in skilled hands the cuff of fabric can be as readily adjusted and

is preferred because it fits any size of arm. The tubing is thick-walled, the rubber reinforced by linen or silk.

The auscultatory method of estimation of the blood-pressure is the most reliable. We owe this method to Korotkoff of Petrograd, and the work of Goodman and Howell in America, and Sir George Oliver in Britain has done much in recent years to draw attention to the valuable information as to the condition of the cardio-vascular system obtainable by study of this somewhat difficult procedure. Any good stethoscope and sphygmomanometer may be used. The latter is placed about the level of the heart, and the stethoscope is held over the brachial artery just above the bend of the elbow and immediately below the lower border of the cuff. The rubber bag of the cuff should be centred over the artery well above the elbow. Pressure by the stethoscope must be light and even lest the vessel be compressed and confusing sounds result. Inflate the armlet until the vessel is so compressed that no sounds are audible ; then slowly and evenly release the pressure by turning the screw-valve attached to the pump. As the column of mercury falls four different tones, or phases of sound, will be heard before there is complete silence. (1) The first phase consists of short, sharp throbs, or beats, and the mercury reading when this first becomes audible indicates the SYSTOLIC BLOOD-PRESSURE. (2) This is succeeded by a murmur type of beat, varying in quality and in duration, but always quite distinct when the heart-muscle is good. (3) The short, sharp beats return, but louder and clearer than during the first phase. (4) The clear tone suddenly becomes muffled, and the mercury reading when this occurs indicates the DIASTOLIC BLOOD-PRESSURE. (5) After this there is silence, sometimes described as the fifth phase. All these phases are easily distinguished. The average reading in 150 young soldiers, aged 23 to 27, in the fourteenth week of training, was : First phase (systolic), 135–125 ; Second phase, 125–104 ; Third phase, 104–80 ; Fourth phase (diastolic), 80–69. It will be noted that the systolic pressure in this group was about 10 mm. above the average normal—a constant feature in subjects undergoing unusual physical effort. Note also the length of the third phase, the phase which, as suggested by Goodman and Howell in 1911, reflects arterial change and effort.

The PULSE PRESSURE is the difference between the systolic and the diastolic pressure, *e.g.* with 120 systolic and 80 diastolic, the pulse pressure = 40. The DIASTOLIC PRESSURE may be regarded as representing the resistance to be overcome by the heart when the aortic valves are opened. The SYSTOLIC PRESSURE indicates the maximum work of the heart. The diastolic is the more constant, more significant and less liable to alter with nervous influence. The systolic reading normally is as three to two of the diastolic ; *e.g.*, S. 120 to D. 80.

The following figures give the average found in several thousand actual readings. The range of the normal limit is not more than fifteen millimetres above or below.

Age 21–30 : 123·5 (*Systolic*), 82·3 (*Diastolic*) ; 31–40 : 125·5 and 85 ; 41–50 : 130 and 86 ; 51–60 : 133·5 and 89 (V. E. SORAPURE).

. **§ 69. Rapid Pulse.**—The rapidity of the heart-beat varies considerably within the range of *health*. In infancy the normal rate is 130, and this may continue in after-life (p. 119). The pulse is normally more rapid during the menstrual period and menopause, in the evenings and after meals. After a severe illness the pulse more easily becomes rapid. The *pathological* causes of quick pulse are numerous. Apart from cardiac affections, or Graves' disease, a quick pulse is relatively unimportant in the young. When the tachycardia is due to *simple causes*, not the result of myocardial changes, the number of the beats falls ten to thirty per minute when the patient alters his position from standing to lying. Exercise, emotion, meals, fever and sleep modify the rate, and the electro-cardiograms are normal. These features differentiate simple tachycardia

from Paroxysmal Tachycardia and Auricular Flutter, in which the pulse-rate is unaffected by posture or the other conditions above mentioned.

1. The *cardiac* disorders in which an increased pulse-rate forms the most striking feature, are fully described in § 59. Paroxysmal tachycardia has special differentiating features. The pulse is quickened also in mitral and aortic regurgitation in the later stages ; it may occur with all forms of degeneration of the cardiac muscle. In extreme dilatation, especially with *auricular fibrillation*, irregularity and rapidity are combined. An *insidious chronic endocarditis*, before the occurrence of a murmur, maÿ be evidenced by tâchycardia as the only symptom for months.

2. Pyrexia is recognised as the most common cause of rapid pulse when the heart is sound.

3. Early *tuberculosis* should be borne in mind as a possible cause of rapid pulse. Other microbic toxins may cause a quick pulse, and should be suspected in cases where the cause is obscure and the condition has continued for some months, *e.g.*, coli infection, streptococcus, staphylococcus, influenza, pneumococcus, and gonococcus. In such cases a course of vaccine treatment usually reduces the rate of the pulse, and the patient's general health improves *pari passu.*

4. Various other *toxic conditions* increase the heart rate. The heart hurry which attends *chronic alcoholism* is very serious, as indicating myocardial degeneration, or neuritis of the vagus. Tobacco first slows the heart, but in large doses paralyses the vagus ; hence excessive smoking may induce tachycardia. Belladonna increases the rate, fulness, and force of the beat ; in toxic doses it paralyses the vagus and produces tachycardia. Tea and coffee may produce temporary heart hurry.

5. Many *affections of the nerves*, functional and organic, are attended by heart hurry, usually transient, but sometimes persistent. Emotional rapidity of the pulse is familiar to everyone.[1] Tumours pressing on the vagus may be evidenced by tachycardia, even if the tumour be small; a rapid pulse may, indeed, be the only symptom.

6. In *Graves' disease* tachycardia is often the earliest symptom.

§ 70. **Slow Pulse,** Bradycardia, is described fully under bradycardia, § 62, where the distinction is given between the slow heart which is pathological and that which is physiological. Napoleon is said to have rarely

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[1] The following severe case of nervous tachycardia may be quoted. In May, 1887, I was hastily summoned to one of the nurses in the Infirmary, who had, two hours before, witnessed for the first time in her life the death of a patient. She was a healthy young woman of twenty-five, in whom there had been previously no manifestations of hysteria. The solemnity of the scene in which she had just played her part was well calculated to have a very powerful emotional effect upon a novice, and she thereupon burst into a flood of tears. From this she recovered sufficiently to play the organ for prayers, but in the middle of the service she was seized with violent palpitation of the heart, accompanied by a pain over the apex, a sensation of " pins and needles " down the arms and legs, and a sense of impending suffocation. I found her in a state of collapse and general tremor, and unable to remember what had happened. The pulse was beating 120 per minute, respiration sighing, and the surface of the body and limbs pale, cold, and covered with a profuse cold perspiration, having previously been, I was told, suffused with marked general redness. There were no physical signs of cardiac or other visceral disease. I administered 30 grains (2) of bromide of potash, 15 grains (1) of chloral, and 3 ℔ (0·2) liq. strychninæ. She gradually rallied, and presently sank to sleep, and the next morning she was herself again.

had a pulse-rate over forty. I knew a gentleman for twenty years, who is now, eighty-two. He never had a pulse-rate over fifty when in health ; he always enjoyed very good health. It is always well to verify an apparently slow pulse by listening to the heart at the same time. In *disease* a slow pulse is of importance chiefly in heart and brain disorders.

1. *In heart disorders* a slow pulse without irregularity is uncommon, but it may be met with in connection with the senile heart, sclerosis of the coronary arteries, atheroma of the aorta, and fatty or other degeneration of the cardiac wall. It is one of the cardinal symptoms of Stokes-Adams disease ; a regular pulse-rate of 30 is practically always due to complete Heart Block (§ 62).

2. If a slow pulse is associated with marked high blood-pressure, *arterial sclerosis* should be suspected, even when no sign of arterial disease can be discovered.

3. Various *gastric derangements* are frequently associated with a slow pulse. This is, perhaps, the commonest cause of slow pulse in children. In chronic dyspepsia slow pulse is usually associated with extrasystoles.

4. Many *nervous disorders* may be associated with slow pulse. Thus :

(i.) If the temperature is raised, the arteries contracted, and the pulse slower than normal, and if with this there be marked irregularity in rhythm, in a child, we probably have to do with an early stage of meningitis.

(ii.) Cerebral tumour is associated not infrequently with slow pulse. It probably occurs only in the late stages, and as a pressure symptom. Halberton mentions a case in which a violent blow on the head was followed by a permanently slow pulse, with syncopal attacks, succeeded by epilepsy. The post-mortem revealed narrowing of the foramen magnum.

(iii.) Various psychical disorders, such as melancholia, general paralysis, and epilepsy.

(iv.) Spinal injuries, especially affecting the medulla and cervical regions.

5. *Drugs*, such as digitalis and strophanthus. Belladonna and tobacco at first may slow the heart.

6. Sometimes in *toxic* conditions, such as diabetes, jaundice, uræmia, and poisoning by carbon monoxide.

7. States of prolonged *exhaustion* and anæmia, and in *convalescence* from acute illness.

§ 71. The **Irregular Pulse** always indicates an altered condition of the heart, and its chief causes are therefore described in §§ 36 and 57, the section largely dealing with those disorders of the heart which may be diagnosed by the features of the pulse. The student is reminded here that the most common cause in young people is *Sinus Arrhythmia* ; in older subjects *Premature Beats*. Here the radial pulse gives a small hurried beat followed by a pause ; on auscultation of the heart the extra beat is audible. The irregularity in which no two beats or intervals are alike, and in which the pulse-rate is usually increased, denotes *Auricular Fibrillation* (§ 61). In the *pulsus alternans* (§ 64) the rhythm of the radial pulse is regular, but there are alternate large and small beats. It is a grave sign when not associated with tachycardia, as it indicates exhaustion of the heart muscle.

In *pulsus paradoxus* there is complete, or almost complete, disappearance of the pulse during inspiration. It is due to either (1) an increase of the " negative " intra-

thoracic pressure which normally takes place at the end of inspiration, or (2) as extreme weakness in the left ventricle, or to both. It can be produced in even healthy persons at the end of inspiration by so contriving that the negative intrathoracic pressure can be *suddenly* increased. It is met with in intrathoracic tumours, pleural effusion, mediastinitis, and adherent pericardium.

The *anacrotic* pulse is a rare condition, resulting from high blood-pressure. The tidal wave is higher than the percussion wave. It is found in some cases of aortic stenosis and aneurysm.

In the *dicrotic* pulse coupled beats may be simulated ; it is recognised by other signs of low blood-pressure and by the fact that the second wave occurs just after the closure of the aortic valves, and is unaccompanied by any heart sound.

§ **72.** **High Blood-Pressure** is, in extreme cases, recognisable by palpating the radial artery ; but the digital method is so unreliable that no attempt should be made to gauge the blood-pressure except by means of an instrument. There are several of these on the market. The best for the consulting-room is probably a modification of Riva Rocci's instrument. There are others which are more portable. The sphygmographic tracing of high blood-pressure is shown in Fig. 27.

The normal blood-pressure varies with age and circumstance. It is rather lower in women than in men, and lower in children than in women.

FIG. 27.—HIGH BLOOD-PRESSURE in a case of Chronic Interstitial nephritis, with Albuminuric Retinitis, under the care of Dr. J. S. Bristowe. The high pressure is indicated graphically by (1) prominence of the tidal wave (unless the arteries are very atheromatous) ; (2) smallness of the dicrotic wave, and its occurrence high up on the diastolic line ; (3) *gradual* sloping of the diastolic line towards the next up-stroke.

The normal variations of pressure at different ages are given in § 68.

The main factors upon which Blood-Pressure depends are three : (1) The force and volume of the heart's output at each systole ; (2) the peripheral resistance to outflow of blood from the arteries ; (3) the elasticity of the vessels themselves. The greater the elasticity of the arteries the more uniform is the pressure and the steadier is the rate of flow between the heart-beats. With a rigid system of tubes the blood flow is inter-mittent and there is a great difference between the systolic and diastolic pressure. The importance of the condition of the vessel wall in the influence of blood-pressure is seen in cases of aortic incompetence. When this abnormality is present a striking difference is always to be found between the systolic blood-pressures in the arm and leg vessels. It is not uncommon to find that the pressure in the leg arteries is 80 to 100 mm. higher than that in those of the arm when the patient is lying down ; and a much greater difference than this is not unusual. If a patient who

presents this phenomenon sits in a hot bath the systolic pressure in the leg vessels will be found to fall till it equals that in the arm, vessels; such a change is dependent upon the alteration in the " tone " of the vessels which is brought about by heat.

The *Symptoms* which accompany high blood-pressure, and which lead us to suspect that condition, are very important, though somewhat variable. They consist of (i.) headache, which may be frontal, occipital, or vertical, accompanied by vertigo from time to time, and a constant feeling of fullness about the head. (ii.) There may be some lassitude, disinclination for exercise, and depression. (iii.) Breathlessness on exertion is common; very often it is paroxysmal, and the patient thinks he has asthma. (iv.) Wakefulness, or sleeping by dozes. (v.) Nose-bleeding, precordial pain, numbness and tingling of the limbs. High blood-pressure is rare in children, but these symptoms occurring in adults, especially in those past middle life, are suspicious, and are confirmed, if upon examination we find the following physical signs : (i.) The high-pressure pulse ; (ii.) on auscultation an accentuated aortic second sound (sometimes accompanied by a reduplicated first sound at the apex) ; (iii.) later on, if the condition persist or frequently return, cardio-vascular hypertrophy supervenes (see Arterial Sclerosis, § 78).

The *Causes* of high blood-pressure are numerous. Among the *predisposing causes* heredity plays a most important part. No age is exempt, but it is more frequent at and past middle life. Males are more subject to the disease, since they are more exposed to the dietetic and other influences which raise blood-pressure. *Exciting* causes bring into operation one or more of three factors—increased peripheral resistance, increased cardiac force, or increase in the volume of the blood. The exciting causes are :

1. Anything in the mode of life which leads to *deficient oxygenation* and *elimination of nitrogenous waste*, such as : Excess of nitrogenous food ; sedentary habits ; constipation. (2) *Toxic conditions* of the blood act probably in the same way. (i.) Chronic renal disease is a familiar antecedent and accompaniment of high blood-pressure. In acute renal disease the pressure rises rapidly, even in children. (ii.) High blood-pressure so frequently accompanies gout that it is known sometimes as the " gouty pulse." (iii.) The diabetes which occurs in persons past middle life is attended by high blood-pressure, but not that more fatal form of diabetes which occurs in younger subjects. (iv.) Plumbism, which is intimately connected with gout and renal disease. (v.) Emphysema, and sometimes other lung conditions (probably by deficient oxygenation). (vi.) Anæmia sometimes. (vii.) Pregnancy. (3) Any condition leading to a *persistent contraction of the arterioles* results in high blood-pressure. It is highly probable that some of the above toxic conditions may act in this way. (4) *Plethora*, by increasing the volume of the blood. (5) *Cardiac hypertrophy*. (6) Polycythæmia, in which there is increased viscosity of the blood. (7) Whenever the heart beats more rapidly and more powerfully—as, for instance, during excitement or during exertion—there must necessarily be a rise of pressure. This is usually transient, but if frequently repeated, as in athletes, it may be a forerunner of a persistent high arterial pressure. (8) In certain *neuro-vascular* diseases there is a tendency to dilatation, and in others to spasm, of the peripheral vessels. In the latter (*e.g.*, Raynaud's disease) there is a frequently recurring tendency to high blood-pressure.

Pathological Effects of High Blood-Pressure and Prognosis.—Temporary high arterial pressure is not serious, but when it constantly recurs or

continues over many months or years, it has grave results. By the physiological law that increased function results in increased growth, there is hypertrophy of the muscular tissues of the whole of the vascular system —that is to say, hypertrophy of the heart and of the muscular tissues of the arteries (Arterial Hypermyotrophy, § 79). This may be said to constitute the first stage. If the increased pressure continue, a degeneration occurs in the muscular tissue of the heart and of the arteries (§ 78). When undue pressure on the arterial system has been in operation for some time, changes take place in the coats of the vessels ; in the larger vessels, such as the aorta, it leads to atheroma ; in the smaller vessels, to sclerosis. Arterial disease of both kinds is usually very patchy in its distribution. Thus, sclerosis of the vessels in the renal area gives rise to granular kidney ; in the hepatic area, to cirrhosis of the liver ; and in the vessels of the brain, to what are known as senile manifestations. The second stage is manifested chiefly by the failure of the heart to compensate for the increased peripheral resistance caused by the rigidity of the arteries. Certain accidents are liable to occur as the result of high blood-pressure, even when this is functional only. Chief amongst these is hæmorrhage. A person with high blood-pressure may bleed from anywhere ; rupture of the arteries into the internal capsule or other parts of the brain, rupture of retinal arteries, hæmatemesis, hæmoptysis, bleeding from the nose, bleeding from the gums, are all common.

The *treatment* of high blood-pressure in its functional stage is one more of general hygiene than of drugs—Rest, physical and mental ; vegetarian and fruit diet ; abstention from tobacco, tea, coffee, cocoa, alcohol. Salines and mercurial purgatives are useful. Vaso-dilator drugs, such as the nitrites, have an effect on the pressure (Fig. 28). If the blood-pressure reaches an unduly high figure, venesection should be practised, and at least 20 ounces of blood withdrawn. Hot baths, Turkish baths, electric light baths, high-frequency currents, and, indeed, anything which will dilate the cutaneous vessels, should be advised, and relaxation exercise is one of the best measures. The iodides of potassium and sodium in large doses produce good effects on the blood-pressure.

§ 73. **Low Blood-Pressure** may be suspected if the pulse, when counted with the patient erect, is rapid, and the rate falls 30 or 40 beats when · the patient is placed in the recumbent posture. As measured by the manometer, 80 millimetres Hg is regarded as an extremely low systolic pressure. To the examining finger the pulse comes up rapidly, rapidly declines, and is very easily obliterated. When the diastolic pressure is very low, and the systolic fairly high, a double wave is felt ; this is known as the *dicrotic pulse*. A sphygmographic tracing shows an increase of the normal depression (aortic notch) before the dicrotic wave, and the dicrotic wave itself is more marked than in a healthy pulse. When the aortic notch falls below the level of the base line, the pulse is said to be *hyperdicrotic* (Fig. 28, e). *Symptoms* of depression, lassitude, prostration, and sometimes dyspepsia and sleeplessness, occur in association with low

arterial pressure.   Purgatives cannot be well borne, and the patient may feel better when the bowels are constipated.   *Capillary pulsation* is sometimes met with in low blood-pressure, particularly with aortic regurgitation.

FIG. 28.—PULSE OF HIGH BLOOD-PRESSURE REDUCED to a state of hyperdicrotism.—Series of tracings showing the efficiency of sodium nitrite in the reduction of high pressure.   Martin R., aged forty-five, chronic parenchymatous nephritis, under the care of Sir William Gairdner in the Western Infirmary, Glasgow.   August, 1895.   *a*, Typical high pressure.   Tracing *b* shows the reduction of pressure after 15 grains sodium nitrite in four doses during the twenty-four hours.   The remedy was continued, and tracings *c* and *d* on successive days show the gradual reduction of pressure effected.   They are normal, excepting for the exaggerated aortic wave.   In the last tracing *e* a condition of HYPERDICROTISM is shown, the high pressure having been entirely replaced by the opposite extreme.   (Kindly supplied by Dr. W. S. Cook, who was the house-physician at the time.)

By drawing a line along the forehead, pressing a glass slide on the mucous membrane of the lip, or lightly pressing down the tip of the nail, the alternate blush and pallor due to the capillary pulsation is well brought

out.   In extreme states a pulse may even be communicated to the veins on the dorsum of the hand.

Causes.—In *health* a persistent state· of low blood-pressure is some-times, though rarely, a hereditary condition.   It may be found also after meals, a warm bath or moist warmth.

1. With *cardiac disease,* in cases of failing heart there is often, but not always low blood-pressure.   The pulse of *aortic regurgitation* is so char-
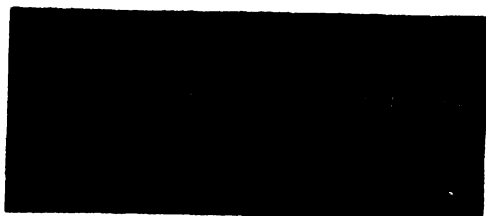
FIG. 29.--LOW BLOOD-PRESSURE PULSE TRACING, from a case of Enteric Fever, showing marked DICROTISM.   Patient aged thirty-seven.

acteristic that the lesion can be diagnosed by it.   It comes suddenly up to the finger, and as suddenly collapses.   This has received the name of " shotty," " slapping," " water-hammer " or " collapsing " pulse, the pulse of unfilled arteries, or *Corrigan's* pulse, after the name of the physician who first described it (Fig. 30).   It is best brought out by feeling the radial while the patient's hand is held up vertically on a level with the head.   Moreover, the artery in aortic regurgitation is large, in contra-

FIG. 30.—WATER-HAMMER PULSE.—Tracing (taken by Dr. Reginald E. Hanson) from a man, aged thirty-four, with typical aortic regurgitation, accompanied by very great cardiac hyper-trophy (bovine heart).   Well-marked capillary pulsation and retinal pulsation were present. Typical collapsing or water-hammer pulse.   He had had anginoid attacks, which were relieved by sodium nitrite.   At the time tracing was taken pulse was 81, respiration 22, and blood-pressure (taken by Hill and Barnard's instrument) 140.

distinction to aortic stenosis, where the artery is small.   The sphyg-mographic tracing of the " water-hammer " pulse shows a long percussion stroke, scarcely any tidal wave, with a sudden down stroke, broken only by a small dicrotic wave.   In cases of a double aortic murmur, this pulse is of great diagnostic significance.   If the pulse has a distinctive " water-hammer " character, the systolic murmur is due not to aortic stenosis, but simply to roughening of the valves or atheroma of the aortic wall.

2. Without organic heart disease low blood-pressure is met with in neurasthenia, in Addison's disease, tuberculosis, and in all *states of ex-*

haustion and debility, such as are caused by over-exertion, physical or mental, deficient and bad food, or anxiety—conditions in which vaso-motor paresis might occur. In its extreme form it is recognised clinically as surgical shock or collapse. In Addison's disease the low pressure is of considerable value in the diagnosis. It is common in adolescents.

4. Low blood-pressure is also met with in all *asthenic varieties of fever*, especially typhoid fever (Fig. 29).

• *Treatment* depends upon the cause. The heart muscle is the important factor to consider. The food should be nourishing and easily digestible. Change of climate is often beneficial. Iron, arsenic, quinine, strychnine, and digitalis are all useful. In collapse warmth should be applied to the surface and pituitary extract 1 in 10 injected intramuscularly.

§ 74. **The Pulse in Relation to Prognosis and Treatment of Disease.**— In acute febrile diseases a full bounding pulse is usual, and its absence warns us that we are in the presence of an asthenic type of case. In fevers the pulse is our chief guide as to whether alcohol should be administered or not. In enteric, if the pulse is weak and the vital powers flagging, stimulants are called for. In chronic affections the pulse is not so valuable an indication of the general condition. In the aged, and, indeed, all persons past middle life, the pulse should be carefully watched from time to time, because a constant high diastolic pressure is the main cause of arterial degeneration. Constant high diastolic pressure may exist with a low systolic pressure, and in time affects the heart (§ 72).

The administration of digitalis and other cardiac remedies is regulated by the condition of the pulse. Thus in cardiac disease digitalis should be given only when the pulse is of low pressure, *quick* or *irregular*. The prognosis of auricular fibrillation may be gauged by the extent of the response to digitalis (§ 61). If, on the other hand, the pulse is coupled, too slow, or if vomiting sets in *during* the administration of digitalis, the drug should be at once withdrawn.

The onset of acute inflammation within the abdomen—needing, perhaps, prompt surgical interference—is indicated by a rising pulse-rate ; anything over one hundred is my own guide. It must be remembered, however, that in some acute abdominal conditions, *e.g.*, perforation of a gastric ulcer, the pulse-rate may not rise for some hours.

The polygraph and the electrocardiograph have revolutionised the methods of prognosis by examination of the pulse, but the clinical observer can still learn by constant and repeated observation much important information by feeling the pulse.

ARTERIAL DISEASE

§ 75. **Symptomatology.**—Among the symptoms to which arterial disease may give rise are giddiness or " dizziness," feelings of faintness, slight syncopal attacks, headache, paroxysmal dyspnœa, gangrene, or

other conditions referable to the extremities (Chapter XVII). But each of these symptoms, excepting the last named, may be caused by disease of some other physiological system. It is only when several of them are met with together, and an examination of the heart and arteries lends confirmation to the idea, that we are led to conclude that the vascular system is at fault. The importance of arterial disease depends more upon its effects on the heart, and indirectly on the other organs and tissues of the body, than upon the vascular condition *per se.*

§ 76. Physical Signs of Disease of the Arteries.—The physical signs are very few in number, and consist simply of a visible or palpable thickening, dilatation, or tortuosity of the superficial vessels, such as the temporals, radials, brachials, and sometimes carotids. There are three features to note concerning the accessible arteries : (i.) The size of the artery should be observed as we compress or roll it beneath the fingers— a feature which sometimes requires considerable experience to recognise. (ii.) The thickness of its wall ; and it must not be forgotten that high arterial pressure may produce the sensation of a thick wall, and *vice versa.* The thickness of the wall is best ascertained by stopping the pulse with the fingers of one hand, and rolling the empty tube beyond the under fingers of the other hand. (iii.) By passing the fingers up and down the length of the tube, the bead-like thickening due to atheroma may be detected.

The arteries are much more prone to disease than are the veins, which is in keeping with the greater liability of disease to attack the left than the right side of the heart—at least, during extra-uterine life. **The Chronic Diseases of the Arteries** which admit of clinical recognition are as follows :

I. Atheroma. II. Arterial Sclerosis (synonyms : Arterial Fibrosis, Arterio-capillary Fibrosis, etc.). III. Arterial Hypermyotrophy. IV. Functional Disease of the Arteries (see Diseases of the Extremities, Chapter XVII.). V. Aneurysmal Dilatation. VI. Chronic Endarteritis, due to syphilis and other causes, is only recognised by its pathological effects (cerebral softening, gangrene, etc., see Diseases of the Nervous System : Anatomy). Acute Endarteritis is generally part of Acute Endocarditis (§ 39). VII. Embolism, or the blocking of an artery by an embolus, is the result usually of cardiac disease, especially infective endocarditis ; or it may be secondary to thrombosis. VIII. Thrombosis, or the coagulation of blood in a living vessel, is usually the result either of local disease involving the vessel, or of some blood change. Both this and Embolism are dealt with elsewhere. See, for example, Localised Dropsy, or Phlebitis (Diseases of the Extremities, Chapter XVII.).

§ 77. Atheroma.—Atheroma has unfortunately come to be used in a somewhat vague sense, but it is taken here to mean a localised or *patchy* thickening of the tunica intima, occurring for the most part in patients past middle age, unaccompanied, as a rule, by any obvious symptoms during life. It starts as a localised hyperplasia in the deeper (external) layer of the tunica intima ; and the change may go on to a fatty, caseous, and sometimes calcareous, degeneration. When it is advanced, the middle, and even the external, coats may be invaded. It is generally more or less wide-spread, but the disease nearly always commences and predominates in the larger vessels—*i.e.*, in the aorta and its branches. Consequently, if it be detected in the

radial or temporal, the inference is that its distribution is extensive and that it involves the vessels of the brain also.[1]

Symptoms are generally altogether wanting.[2] However, when the disease involves the aorta (and it nearly always commences in that situation), it impairs the elasticity of that structure, and gives rise to an accentuated second sound of the heart in the aortic area.   In the more advanced cases atheroma may sometimes be detected as a nodular or beaded thickening in the radial, temporal, and other superficial arteries, which can be felt by moving the fingers up and down the artery. The patient is, however, unaware of its existence, and may live to old age, unless he be subject to high tension or other cardio-vascular disorder.

Consequences.—Hæmorrhage is apt to occur in these cases, due not to the atheroma, but to thinning and dilatation of the vessel on the proximal or distal side.   Occurring, as it does, only in old people, it is accompanied by, and possibly aids in the production of, debility and other signs of old age ; but it is wonderful how extremely common widespread patchy atheroma is even in healthy old people who die of pneumonia or some other intercurrent malady, e.g., the woman aged one hundred (see footnote 2, below).

No *treatment* will remove the atheromatous condition, but its presence is one of the indications for the avoidance of the causes of high blood-pressure.

§ 78. **Arterial Sclerosis.**—The term " arterial sclerosis " is here used in an anatomical sense, as a generic term to mean any widespread thickening and hardening of the arterial coats which leads to loss of elasticity and contractility of the arteries which is *clinically recognisable.* It is too often forgotten that what may be called the " parenchyma " of the arterial system—that is to say, its functionally active part—is the middle or muscular coat. Upon the proper functioning of this coat depends the whole of the regulator mechanism of the arterial system. My experience at the Paddington Infirmary, which offered a very extensive field for researches into the pathology of the vascular system, went to show that, although very wide changes might exist in the tunica intima or tunica adventitia without symptoms, very slight changes in the media were nearly always attended by some kind of symptom or effect during life. Histologically, therefore, I differentiate intimal, adventitial, and medial sclerosis—the last named being by far the most important.[3] The subject of arterial disease is surrounded with considerable obscurity, partly because the same words are used by different observers in very different senses, and partly on account of the difficulty of making a sufficiently exhaustive examination of the entire arterial system in any given case. A dozen sections of the liver, for instance, will give a fair idea of a morbid change in that organ ; but to form an accurate conception of the structural changes in the arterial system in any given case, it may be necessary to carefully examine one or two hundred sections, taken from many different vessels, and to make very accurate *measurements* of these, both naked eye and microscopic.

. [1] Some observers say that, with the exception of the coronary arteries and the vessels of the brain, it does not usually affect the smaller arteries, but I have satisfied myself of its existence in the radials and nearly all the arteries of that size in the body in advanced cases of atheroma. ͵

. [2] This absence of symptoms has sometimes led me to conjecture whether atheroma might not be, in a sense, a conservative process, an idea which is further strengthened by two other important circumstances—viz., (1) That the change was most constantly found in those situations exposed to the shock of the systole, as, for instance, on the upper aspect of the arch of the aorta and in the angle of the bifurcation of arteries. (2) It was a constant—and therefore, one might say, a physiological—change in greater or less degree in *all* elderly people dying in the Infirmary, no matter what might be the cause of death.   One of the very notable cases was that of a woman, aged one hundred, who died of pneumonia, and who had not at any time presented any cardio-vascular symptoms during life.

[3] " On Arterial Sclerosis " (Transactions of the Pathological Society of London, 1904).

Symptoms.—The clinical course of the disease may be divided into two stages : (*a*) That in which the ventricular hypertrophy is sufficient to compensate for the increased peripheral resistance ; and (*b*) that in which the left ventricle begins to fail—that is, to yield and dilate.

(*a*) In the first stage (1) the patient may be free for many years from any symptoms referable to the vascular system, so long as the increased peripheral resistance is adequately and not excessively compensated for by the ventricular hypertrophy. Sometimes the patient may come to us for loss of vigour dr breathlessness ; but more often the. thickened vessels are discovered, so to speak, by accident, when the patient comes under our notice for some other malady. On examination, however, we may find that the arteries are visibly and palpably thickened at the wrist and on the temples, being cord-like, and sometimes elongated and tortuous. The feel of the vessel much resembles that of high blood-pressure (a condition, by the way, which may coexist in the earlier stages), but by compressing the pulse above and so emptying the vessel, and then feeling the artery beyond, the thickening of the wall may be readily revealed. By sliding the fingers up and down, it may be distinguished from the beading of atheroma. (2) As the disorder progresses, the patient loses his former vigour, mental and physical. He is, in a word, " old before his time," for it has been truly said a man is " as old as his arteries." This loss of vigour is no doubt due to the fact that all the organs and tissues are deprived of that regulation of nutrition which depends upon the elasticity and healthy contractility and relaxation of the muscular tissue of the arteries. (3) Breathlessness, and a tendency to rapidity of pulse after slight, or even without, exertion are, in my experience, the next most constant features. Sometimes the dyspnœa is paroxysmal,

FIG. 131.—ARTERIAL SCLEROSIS.-·Section of a radial artery magnified about 400 diameters, stained with acid orcein to show granular degeneration of muscle fibres in the middle coat, which commences and *predominates in the internal layers of middle coat.* This method of preparation brings out the elastic tissue, as may be seen in tunica adventitia and tunica intima. Similar sections stained sufficiently long in logwood brought out the rod-shaped nuclei of the muscle fibres, showing that the change is not a fibrosis, as Gull and Sutton maintained. The appearances described by them as arterio-capillary fibrosis can always be produced by prolonged hardening coupled with insufficient staining with logwood.

and the case resembles asthma. The heart is irritable, and this is evidenced clinically by breathlessness and a pulse which easily becomes rapid and irregular.

(*b*) When the second stage is reached, symptoms arise which, as a whole, form a distinct and well-marked clinical picture. They are due partly to failure of the arterial functions, but mainly to the failure of cardiac compensation. In addition to the preceding, which become emphasised, the symptoms are as follows : (1) Vertigo may have occurred before the second stage is reached, but the attacks now become more frequent. Indeed, about nine-tenths of the cases of senile vertigo met with in the infirmary could be traced to this malady. It is not, however, a true vertigo, but rather a sensation of " swimming in the head " or " dizziness," and the patient either feels as if he were " going to fall " or " going to faint." So frequently did these attacks occur among the aged and so-called " healthy " old people in the workhouse that they used to pay but little attention to their frequent falls due to this cause. (2) Actual fainting attacks are met with less frequently, but when present they are of more serious import. They vary from a slight interruption of the continuity of thought on the one hand to a prolonged faint or epileptiform seizure on the other. (3) Physical signs pointing to a varying degree of hypertrophy and dilatation of the heart are revealed on examination, together with accentuation of the second sound over the aortic cartilage, if high blood-pressure be present. (4) Anginoid attacks, or true angina pectoris, form another symptom. Quite three-fourths of the cases of angina pectoris met with in the infirmary were attended by this condition. (5) In extreme states of the disease, especially when accompanied by atheroma or endarderitis, there may be gangrene of the extremities, cerebral softening, either localised or diffuse, and similar changes in other viscera. Miliary aneurysms may arise in the periphery of the arteries of the brain, and hæmorrhage in this situation is one of the most frequent consequences. There is always in these cases a tendency to the development of low forms of inflammation, especially " senile pneumonia." (6) Chronic interstitial nephritis is frequently, but not necessarily, associated with arterial sclerosis. Some hold that it is essentially a generalised arterio-renal disease, and that, although arterial thickening may occur without renal disease, the latter is always attended by more or less arterial change.

• In the *Etiology* (1) heredity is certainly one of the most important factors, and families are found in which every member shows a tendency to this disease on reaching a certain age. (2) As regards age, the disease is usually met with only in the latter half of life [1] ; and it is rather more frequent among men. (3) The pathology of the complaint is not yet worked out, but in many cases careful inquiry will reveal one or more of

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[1] Syphilitic arterial disease is a patchy endarteritis which occurs in younger subjects, usually more or less localised, and therefore does not come within the scope of our opening definition. •

the causes of a constant high blood-pressure (q.v.). Various toxic blood
states possibly act in this way, and there is often a history of alcohol,
lead, or gout. (4) Overwork, and physical strain by leading to an over-
filling of the arteries, are also possible causes, on the principle that increased
function causes first increase of structure, and, later, degeneration of the
muscular coat.

Diagnosis.—Arterial sclerosis may have to be diagnosed from (1) high
blood-pressure, by stopping the blood current and examining the artery
beyond ; (2) atheroma which gives to the vessel an unequal or beaded
character. (3) Granular kidney, in its slow, insidious onset and vague
symptoms, closely resembles arterial sclerosis in its clinical history, and
can only be distinguished from it by the presence of urinary changes.
The arterial and the renal changes are frequently associated. (4) Other
causes of progressive debility (Chapter XVI) may have to be distinguished
from arterial sclerosis.

Prognosis.—In the first stage, though nothing can be done to abolish
the thickening of the arterial walls, much may be done to prevent its
advance, and if the patient escape pneumonia and other inflammatory
conditions to which he is liable, he may live many years. The whole
question of prognosis turns very largely on the state of the heart. If
the breathlessness is considerable, and the physical signs show marked
cardiac dilatation and the pulse is irregular and rapid, the patient is
not likely to live more than a year or two. If, on the other hand, cardiac
compensation is good and the patient feels but little distress on movement,
then the outlook is not unfavourable.

Treatment.—The indications for treatment are (a) to keep the blood-
pressure low ; (b) to aid the heart ; and (c) to avoid any extra strain
being thrown upon the heart or vessels. (1) The blood-pressure is reduced
by avoiding the causal factors. High blood-pressure, *per se*, does not
require to be reduced if the patient complains of no symptoms. Rapid
reduction by drugs or electricity is not permanent, and may be dangerous,
if the cause is left untreated. Hygienic measures are of great value, and
the patient's duration of life will depend upon the kind of existence he
can afford to live. He should live a very regular life, free from any
strain on mind or body. The diet should be strictly moderate, especially
as regards proteids, and should be readily digestible. The toxins arising
from the digestive tract must be carefully considered in each case, and
the diet altered according to the individual indications. Elimination by
bowels, kidneys and skin must be assisted. Unless heart failure is very
pronounced, alcohol should be avoided. Where cardiac failure has set
in, cardiac tonics and other remedies may be administered on general
lines (see Cardiac Failure, § 56). The question of baths and passive
exercises is a most important one, and while some advocate them
strongly, others say that they are attended with considerable danger
by increasing the tendency to hæmorrhage and the other consequences
above referred to; but, in my belief, if the arterial disease is not very

advanced, the heart undoubtedly derives considerable benefit by this treatment.

§ 79. **Arterial Hypermyotrophy** is a term which has been employed by the author to imply a generalised increase in the muscular tissue of the arteries. In a paper read before the British Medical Association at Bournemouth, in 1891,[1] based upon a collection of cases which were observed in the Paddington Infirmary, it was shown that a generalised increase in the muscular tissue of the arteries occurred as a distinct clinical and pathological entity, consequent, in all probability, on states of prolonged or frequently recurring high blood-pressure, by the simple physiological law (that increased function leads to increased structure. In 1895 Drs. Dickinson and Rolleston [2] showed that a widespread increase of the muscular tissue of the arteries occurs throughout the body in some cases of renal disease. Sir Clifford Allbutt [3] has described under the name Hyperpiesis (persistent high blood-pressure and its attendant symptoms), a condition which, from a clinical standpoint (for none of the cases were confirmed by autopsy), probably corresponds with the condition which the writer, from an anatomo-clinical standpoint, has called " arterial hypermyotrophy."

The change itself consists of a hypertrophy of the middle or muscular coat of the arterial wall. It affects principally the medium and small-sized arteries of the body —those which normally contain more of this tissue than is found in the larger vessels. Patients may exhibit no symptoms, and rarely die in the early stages of the disease or until some granular or other degeneration has taken place in the hypertrophied tunica media. The condition, however, is by no means an infrequent one, to judge from the records of the Paddington Infirmary, and if it were not such a laborious task to examine the arteries of the body, more cases would doubtless be revealed.[4]

Symptoms.—(1) The arteries have a thickened but elastic feel, although they may be of normal size. In a few cases the author has been able to confirm this by means of Oliver's arteriometer. The prolonged first stage is always accompanied sooner or later by cardiac hypertrophy. It may exist unknown to the patient for many years, and be overlooked by the doctor, or, like arterial sclerosis, be discovered accidentally. Sooner or later, however, one or more of these symptoms arise, viz., (2) *postural vertigo* from loss of arterial adaptability to posture ; (3) dyspnœa (sometimes of an asthmatic or paroxysmal character) ; (4) persistent or recurrent headache ; and (5) symptoms of high blood-pressure. (6) In the second stage of the disease, when granular degeneration and consequent rigidity are present, the symptoms are indistinguishable from those of arterial sclerosis, which, in point of fact, supervenes. It is more often found in persons over forty.

Effects.—The results of the thickening in the *first* stage are (i.) a diminution of the lumen of the vessels by reason of the tonic spasm ; (ii.) a more or less permanent

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[1] Cases of arterial hypermyotrophy and the resulting degenerations in the muscular tissue were published in the *British Medical Journal*, January 23, 1897, and the Transactions of the Pathological Society of London, 1904.

[2] See the *Lancet*, 1895, vol. ii., p. 137.

[3] The Lane Lectures, *Philadelphia Medical Journal*, April, 1900, pp. 400–500 ; and elsewhere (*e.g.*, " The Hunterian Oration," *c.* 1885).

[4] It has been said in criticism of these observations that the change consists of a swelling of the individual muscular fibres rather than a numerical increase. This is difficult to refute, because the opportunity does not often occur of examining the arteries before the granular swelling—which is also a consequence of the same cause (high blood-pressure)—has also occurred. But, in the first place, I would point to the actual occurrence of true hypertrophy in renal cases, as shown by Rolleston and Dickinson.[2] Secondly, I have occasionally been fortunate enough to secure cases—dying by accident, for example—which undoubtedly exhibited a true hypertrophy without degeneration. Thirdly, a very careful examination of several of my cases shows that there was an actual increase in the unstriped fibres, in addition to their degeneration ; and, fourthly, Sir Clifford Allbutt's *clinical* observations undoubtedly lend confirmation to the existence of such a pathological condition as arterial hypermyotrophy,

increase of the blood-pressure; (iii.) *pari passu* with the arterial thickening and high blood-pressure there is hypertrophy of the left ventricle. As the result of the insufficient or ill-regulated blood-supply, the tissues are insufficiently nourished, and tend to degenerate, and are more readily prone to inflammation and disease. The patient loses his mental and bodily vigour. In the *second* stage cardiac compensation fails, and the middle coat of the arteries degenerates—the consequences of which are identical with arterial sclerosis above described. Arterial hypermyotrophy is no doubt often associated with granular kidney, probably in about half the cases. But from the cases which the author has collected, it is evident that arterial hypermyotrophy may occur quite independently of renal disease of any kind.

The *Prognosis* of the condition in its early stage is favourable, if the patient can live a careful life, although its existence adds to the gravity of intercurrent diseases.

The *Treatment* in the early stage is that of high blood-pressure (§ 72); in the later stages that of arterial sclerosis (§ 78). Symptomatic treatment is always useful. For the breathlessness, nux vomica and digitalis, and aperients, especially 1 or 2 grains (0·06–0·013) of calomel, are at all times useful. For the attacks, especially the vertiginous attacks, nothing gives so much relief as nitroglycerine, and for the severe ones, occasional inhalations of amyl nitrite. This is an undoubted fact, and constitutes one of the reasons in support of the theory that these seizures are of entirely circulatory origin. It also supports the idea that it is muscular spasm, and not the degenerated arterial wall, which produces the vertigo. For the fainting attacks, alcohol is indicated in small doses, but I have found that alcohol in anything but very small quantities aggravates the symptoms and consequences of the disorder.

§ 80. **Functional Diseases of the Arteries.**—Of functional diseases or vaso-motor derangements we know but little, although several very important maladies are attributed to this cause—*e.g.*, Raynaud's disease and migraine. Functional derangement of the arteries is also manifested by a large number of symptoms, many of which are vague and evident only to the patient. On this account they are apt to be regarded by medical men as unimportant, and it is true that they are not serious in the sense of being lethal; but to the patient they are often extremely disagreeable, irksome, and often terrifying. Of such we may mention alternate flushing and pallor ("flush-storms"), dead hands, cold hands and feet, chilblains, various other erythematous conditions, blue nose, palpitation, tachycardia (§ 59), paroxysms of copious urination, acroparæsthesia, erythromelalgia, feelings of suffocation, pseudo and true angina pectoris, feelings of tingling, itching, throbbing, and actual swelling of the limbs. A case of the last named is referred to under the heading Œdema (§ 455).

Syphilis affects the arteries in two ways : (1) a proliferation of the intima of small vessels reduces their lumen and interferes with the nutrition of the parts supplied by these vessels. This condition also predisposes to thrombosis in the affected vessel, and explains the occurrence of many cases of cerebral thrombosis. (2) A weakening of the muscular coats of the large vessels as seen typically in syphilitic mesaortitis and brought about probably by obliterative changes in the vasa vasorum. (3) A proliferation of the intima of the large vessels, especially of the root of the aorta, is commonly associated with mesaortitis. It may lead to extensive scarring of the intima. The condition is often associated with anginal pain.

Aneurysmal Dilatation of the Arteries belongs to surgery, excepting aneurysm of the thoracic aorta (see § 65), the abdominal aorta (§ 191), and the cerebral arteries (Chapter XIX). **Embolism** and **Thrombosis** are referred to under Diseases of the Extremities (Chapter XVII) and the Brain (Chapter XIX).

CHAPTER VI

THE LUNGS AND PLEURÆ

Owing to the extreme vascularity of the lungs, it is not surprising that inflammation of these organs is a frequent complication of acute general or blood diseases. Thus, inflammation of the lungs is one of the commonest accompaniments of the acute specific fevers and other microbic disorders. Again, in the generalised blood infection which arises from a local tuberculous focus, the lungs are, as we should expect, frequently the seat of tuberculous lesions, and 'there are three *acute* forms of tuberculosis in which the lungs are more or less involved—namely, a tuberculous form of Acute Pneumonia (§ 101), Acute Miliary (generalised) Tuberculosis (§ 411), and Acute Pulmonary Tuberculosis (§ 96). All the blood of the body is oxygenated in its passage through the lungs, and the lungs are in consequence the great defensive organs of the body. Close, heated rooms with tainted air not only prevent the lungs from duly performing their defensive function, and hence decrease the power of the blood to cope with disease, but they are also the means of conveying disease to the lungs themselves. Chronic pulmonary tuberculosis (phthisis), one of the scourges of civilization, is due to the inhalation of air containing tubercle bacilli. Both for the protection of the lungs themselves and of the entire organism which demands a pure blood-supply, the importance of the gospel of fresh air cannot be overrated.

PART A. SYMPTOMATOLOGY.

The **Cardinal Symptoms** of diseases of the lungs are **cough, breathlessness, expectoration,** and sometimes **pain in the chest** and **hæmoptysis.** The more general symptoms are pyrexia, emaciation, and debility. The heart, more especially the right side, suffers sooner or later in all serious or prolonged pulmonary diseases by interference with the pulmonary circulation.

§ 81. Concerning **Cough,** if it is attended by expectoration (as in 1 to 4 below) it points to definite changes either in the lungs or throat. If without expectoration (as in 5 to 8 below), it may point to simple congestion of the throat or larynx, to the presence of pleurisy, to the early stage of some pulmonary disorder, or to some source of reflex irritation. The *Causes of Cough* are as follows :

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1. The commonest form of cough is that recurring WHEEZY cough, attended by expectoration, so typical of bronchitis.

2. PAROXYSMS of coughing followed by vomiting occur in *whooping-cough* and advanced *phthisis*. *Bronchiectasis* is attended by paroxysmal cough with fœtid expectoration at intervals, so also is the rupture of an empyema or liver abscess into a bronchus. Paroxysmal cough, with or without expectoration, occurs with enlarged bronchial glands and other *mediastinal tumours*.

3. The HAWKING cough of throat affections is very characteristic, and is met with in *catarrhal pharyngitis*. It also occurs in *nervous* subjects. It is also associated with digestive disorders, where there is often a collection of mucus in the pharynx with chronic liver disease and with thyroid enlargements.

4. The IRRITABLE cough, most marked in the early morning and on going to bed, is especially associated with *early phthisis*. There may or may not be much expectoration.

5. A NIGHT cough may be due to a *long uvula*. When a patient complains that a cough is worse at night or on lying down, the uvula should be carefully examined. A slight degree of congestion will cause considerable elongation of the uvula, so that it will irritate the back of the pharnyx when the recumbent posture is assumed. A night cough is also associated sometimes with *threadworms* in children.

6. The long BARKING or nervous cough of *hysteria* is very characteristic. It is unattended with expectoration.

7. The SHORT SUPPRESSED cough associated with *pleurisy* or *pleuro-pneumonia* is so characteristic as to be diagnostic ; in the former it is unattended by expectoration.

8. The GANDER OR BRASSY cough (due to laryngeal paralysis) associated with aneurysm and other mediastinal tumours is typical, and when once heard is readily recognised.

9. The REFLEX cough, due to irritation in the area of the pneumo-gastric, may be caused by (i.) *gastro-intestinal* disorders, such as dyspepsia, constipation, diarrhœa, or worms in children ; (ii.) *pericarditis ;* (iii.) *carious teeth ;* and (iv.) *ear* troubles, such as impacted wax ; (v.) *abdominal* disease in which there is irritation of the diaphragm—*e.g.*, by subphrenic or liver abscess.

The *Diagnosis* of these varieties of cough is somewhat important in practice, since they arise from, and may be met with, in affections other than those of the lungs. When a short dry cough is set up by going into the cold, it may be due to pharyngeal congestion or irritation. In simple throat affections the cough comes on in paroxysms, especially after talking. On the other hand, if such a cough comes on in a warm atmosphere, we should suspect phthisis. In chronic irritation of the larynx or trachea the cough is worst in the early morning, when a paroxysm is induced by the effort to bring up a little glairy mucus. The face is congested, there is difficult inspiration, even vomiting.

The *Treatment* of cough depends upon the cause, but, in general terms, irritable coughs may be soothed by bromides, minute doses of opium, heroin $\frac{1}{12}$ to $\frac{1}{6}$ gr. (0·006–0·01), or codeia gr. $\frac{1}{4}$ (0·03), by a linctus of squills and tolu, or by various medicated lozenges, such as the B. P. morphia and ipecacuanha and krameria lozenges.

§ 82. Breathlessness, or dyspnœa, is another symptom of lung affections. The causes of breathlessness are dealt with in more detail in the symptomatology of cardiac disorders (§ 20). The types of breathlessness special to respiratory disorders are :

1. Breathlessness attended by SNIFFING and NASAL BUBBLING is caused by *nasal* or *naso-pharyngeal catarrh*. The obstruction in the nose or mouth usual in such a condition may also cause considerable stertor at night-time.

2. STRIDULOUS respiration, in which the stridor attends both inspiration and expiration, is caused by obstruction in, or *pressure upon*, the trachea or larynx. It is accompanied in severe cases by drawing in of the epigastrium and lower costal cartilages during inspiration (§§ 145, 150, and 151).

3. Dyspnœa attended by considerable WHEEZING or rhonchi in the chest is very characteristic of *bronchitis*, attended usually by emphysema.

4. The LABOURED respiration which attends other *gross diseases* of the lungs is different from any of the foregoing. Under this heading also comes the expiratory dyspnœa of *emphysema*, which is due to the fixation of the chest in a position of inspiration. Undoubtedly the commonest lung condition giving rise to dyspnœa is emphysema, which is revealed by a barrel-shaped chest and hyper-resonance.

5. A rapid respiration with altered PULSE-RESPIRATION RATIO is almost diagnostic of lobar pneumonia. In children there is seen in this disease a characteristic working of the alæ nasi.

6. PAROXYSMAL dyspnœa is present in asthmatic attacks, but is more often an indication of *cardiac disorder* (§ 21).

§ 83. Pain in the Chest is usually present with affections of the pleura, but otherwise it is not a constant symptom in pulmonary disorders. The various causes of pain in the chest are enumerated in § 25. The following are the chief types of pain met with in diseases of the lungs :

(i.) The SHARP, cutting, stitch-like pain of *pleurisy*, before the effusion separates the inflamed surfaces, is greatly aggravated by drawing a long breath. This is undoubtedly the commonest of the pulmonary causes of pain in the chest, and this symptom in *pneumonia* indicates involvement of the pleura. It must be remembered, however, that in some *sub-diaphragmatic diseases*—e.g., of the liver, spleen, or colon—pain is also felt on deep inspiration. One of the most intense forms of pain in the chest is due to *diaphragmatic pleurisy*. It is referred along the lower costal margin, and is accompanied by very shallow respirations, which are chiefly or entirely thoracic. Hiccough is occasionally associated. (ii.) A SORENESS behind the upper part of the sternum attends the onset of *acute bronchitis*. (iii.) SUDDEN severe pain, followed by considerable pulmonary and general distress, occurs with the onset of *pneumothorax*. (iv.) SUDDEN pain, attended by hæmoptysis, marks the occurrence of *embolism* of the lung or rupture of an aneurysm into the lung. (v.) *Cancer* of the lung may or may not be accompanied by pain, according to its proximity to the

pleura or other sensitive structures.   (vi.) All *mediastinal tumours give rise sooner or later to pain in the chest.*

The presence of **expectoration** or sputum is an important sign ; its physical appearance may lead to the diagnosis of certain lung diseases. It must be examined by the physician, and it is therefore described in § 91. It must be remembered that children usually swallow sputum ; so also adults with bad habits or unconsciousness.   Expectoration from the pharynx must not be mistaken for expectoration from the bronchi or lungs. The amount of coughing required to void the sputum may aid diagnosis—*e.g.*, in the early stages of bronchitis much coughing brings up a little tenacious sputum, in the later stages moderate coughing brings up much frothy muco-purulent sputum.

§ 84. **Hæmoptysis** means the spitting of blood (*αἷμα*, blood ; *πτύω*, I spit), but the term is confined to the expectoration of blood from the organs of respiration.

The *fallacies* with regard to this symptom are very important, and it is sometimes as difficult as it is important to decide whether the blood comes from the throat or nose, from the stomach, or from the lungs.   For purposes of malingering, the condition has been simulated by means of friction of the gums.   The differentiation is given more fully under Hæmatemesis (§ 216), but it may be mentioned here that blood coming from the lungs is thus characterised : (i.) It is preceded and accompanied by a tickling cough (if the blood be large in quantity it may excite retching on touching the pharynx) ; (ii.) the patient usually goes on *coughing* up flecks of blood for some time afterwards ; (iii.) the blood has a bright red colour, is alkaline, and frothy (if very profuse, it may be dark in colour and without froth) ; (iv.) physical signs of disease of the lungs are usually, though not always, present—they may be absent in the early hæmoptysis of phthisis ; (v.) the antecedent history of the patient may point to pulmonary tuberculosis or to cardiac disease, these being undoubtedly the most common causes of hæmoptysis.

Causes.—For practical purposes the causes of hæmoptysis may be divided into two groups :

(*a*) Those which produce slight and sometimes protracted or recurrent bleeding ; and (*b*) those which produce a copious bleeding at one time.

(*a*) *Causes of Slight and Sometimes Protracted Hæmoptysis.*—I. PHTHISIS is by far the commonest cause. The hæmoptysis of phthisis may occur either in the early or in the advanced stage of the disease, and in *either* case it may be small or very large in amount. The presence of this cause may be recognised (i.) by the previous and family history of the patient ; and (ii.) by evidences of congestion, consolidation or cavitation of the lung (§ 107). Nevertheless, the most careful examination may fail to reveal any signs, because hæmoptysis is frequently the earliest symptom of invasion by the tubercle bacillus.

II. CARDIAC DISEASE, especially mitral stenosis or the late stage of

mitral regurgitation, is the next most common cause of hæmoptysis. It may arise in such cases either from congestion, or, more rarely, embolism of the lungs. In both cases evidences of cardiac disease are present.

III. Various PULMONARY DISEASES other than phthisis may be attended by slight hæmoptysis. Thus, in *acute bronchitis* the sputum may contain streaks of blood from time to time ; and in *pneumonia* the sputum is rust-coloured about the third or fourth day of the illness. In *chronic bronchitis with emphysema* the sputum may at times be blood-streaked. The hæmoptysis due to primary or secondary *malignant disease* of the lung may be recognised by the irregularity of the physical signs. *Gangrene, abscess,* sporotrichosis and other *fungi* infections, and *hydatid* may cause bleeding. Distoma pulmonale is the cause of so-called *endemic hæmoptysis* in Japan.

IV. ULCERATION or Nævi of the upper part of the respiratory passages may give rise to hæmoptysis, small in amount, and apt to be recurrent. A careful examination of the throat and larynx generally reveals this cause.

V. Erythæmia, purpura, hæmophilia, scurvy, leucocythæmia, and some other BLOOD CONDITIONS may be attended by bleeding from the lungs. These causes are for the most part rare, but when present are readily recognised. It may also occur with the eruptive fevers.

VI. VICARIOUS MENSTRUATION as a cause of hæmoptysis is disputed by some. It is recognised by its occurrence at the time when menstruation is due, the normal menstrual function being absent, and by the absence of signs of disease in the lungs.

VII. CONSTITUTIONAL or idiopathic causes. There are certain patients in whom slight hæmoptysis occurs from time to time, the history and examination revealing nothing, and the patient living to a good old age. The hæmoptysis in such cases is explicable by two hypotheses—the presence of undiscoverable tuberculosis, or a transient congestion due to some constitutional cause. Thus Sir Andrew Clark found hæmoptysis in subjects of the arthritic diathesis. It occurs in subjects of arterial and renal disease.

§ 85. **Pulmonary Embolism** complicates mitral disease, septic venous thrombosis, and other conditions in which there is *intra-vitam* clotting in the vessels. Small emboli may give rise to few clinical signs, the chief being pain in the chest, sudden dyspnœa and hæmoptysis. The appropriate *treatment* is the administration of cardiac and general stimulus—*e.g.,* strychnine, digitalis, ether.

When large thrombi are dislodged from distant parts and travel to the lung, the patient dies at the moment of their impaction in the pulmonary arteries. There are no premonitory symptoms ; even the existence of a clot may be unsuspected. Such clots are not uncommonly found to have originated in the pelvic veins. Cases of sudden death following operations when the patient appeared to be in excellent condition are usually due to this variety of thrombosis. For these cases there is no *treatment* except a watchful care lest patients are allowed to get up too soon after operations.

(b) *Causes of Hæmoptysis in which there is a Considerable Quantity of Blood at One Time.—*I. PHTHISIS.—Copious bleeding (which may be continuous, perhaps for hours or for a day or two), without ending fatally, is almost invariably due to pulmonary tuberculosis. The chief features by which it is recognised are given above.

II. Rupture of an ANEURYSM into the trachea or bronchus is a by

no means rare accident in the history of that malady. It is the one cause of hæmoptysis which is usually followed by immediate death, though in some cases there may be a considerable leakage going on for a day or two before the final issue (§ 65).

III. ULCERATION of the larynx, throat, trachea, though usually causing small and recurrent hæmorrhages, occasionally leads to a large amount of hæmorrhage.

Differentiation.— In order to arrive at a diagnosis of the cause of hæmoptysis in any given case, we must *first* of all inquire into the patient's history ; *secondly,* examine the chest (lungs and heart) very thoroughly ; and, *thirdly,* use the laryngoscope to investigate the larynx and naso-pharyngeal passages.

The *Prognosis* depends, of course, upon the cause. Hæmoptysis is nearly always a serious symptom, and when profuse is followed by considerable debility. In this way it may hasten the end of an advanced case of phthisis. But the hæmoptysis of early phthisis, though indicating definite involvement of the lung tissue, is not so serious, and with proper precautions the patient may completely recover and live to old age.

Treatment.—(a) For *profuse hæmorrhage* immediate treatment is necessary. The patient must be kept lying flat and at absolute rest in bed. Ice is usually applied to the chest, but it should not be kept on in one place longer than twenty minutes at a time. The nourishment allowed must be cold. A hypodermic injection of morphine, gr. ⅓ (0·02), or full doses of opium with acid. sulph. dil., or turpentine internally (℩ xxx. (2) in mucilage four-hourly, and gradually reduced), are the most efficacious remedial drugs for immediate administration. Ac. sulph. dil. ℩ x. (0·6) with alum, gr. v. (0·3), may then be given every twenty minutes. Whitla recommends turpentine vapour in the room. Other drugs recommended are hypodermic injections of pituitrin, atropin, or digitalin ; amyl nitrite inhalations in the hope of relieving the veins and capillaries by dilating the arteries ; and gallic acid or hazeline by the mouth : all these measures are of very doubtful benefit and in most cases are more likely to do harm than good. In expert hands, artificial pneumothorax gives excellent results.

(b) When hæmoptysis occurs in *small quantity,* calcium chloride gr. xx. (1·2) every four hours, for six or eight doses, is said to render the blood more coagulable. The hæmorrhage of congestion due to cardiac disease should not be checked, unless it becomes excessive, as it relieves the pulmonary congestion. When hæmoptysis occurs in elderly arthritics, give a sedative cough mixture, saline purgatives, iodides, and cod-liver oil.

PART B. PHYSICAL EXAMINATION.

The physical examination of the lungs is carried out by means of Inspection and Mensuration, Palpation, Percussion, and Auscultation.

§ 86. Inspection and Mensuration.—The inspection of the chest must be carried out in a good light, and the patient must be instructed to stand

or sit erect, or, if in bed, to lie flat and evenly, and to breathe deeply. After noting the movements from the front, examine the back, then look from behind over the clavicles in order to make out the slighter distortions or inequalities of the chest. By inspection and mensuration we note (1) the shape and size of the chest ; (2) the rate and character of the breathing ; (3) the chest capacity. The chief landmarks of the chest are mentioned in § 33, and the regions into which, for descriptive purposes, it is divided anteriorly, are given in Fig. 32. Posteriorly the chest is divided into the suprascapular, scapular, and infrascapular. The scapular region is divided, by the scapular spine, into the infra- and supra-
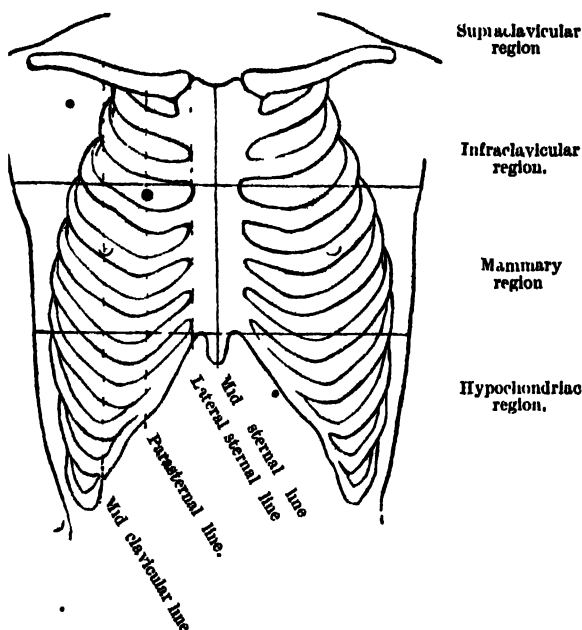
Fig 32 —Anterior Thoracic Regions

spinous regions. The names sufficiently indicate the positions of the various regions.

1. *Rate and Character of the Breathing.* -The rate varies normally from 15 to 20 per minute, or one-fourth the rate of the pulse ; and any change in this proportion, or pulse-respiration ratio, should be observed. Notice whether the breathing is rapid, slow, shallow, or irregular. The respiration should be counted without the patient's knowledge ; thus while counting the breathing, it is a good plan to feel the radial artery as if you were examining the pulse. Both sides should move equally. Flattening or immobility of any part of the chest points to disuse of that part of the

lung—*e.g.*, from consolidation. Flattening or protrusion of the inter-spaces indicates fluid. Drawing in of the interspaces on both sides during inspiration is indicative of some interference with the free entry of air into the lungs (inspiratory dyspnœa), as in diphtheria or other cause of obstruction of the larynx or trachea. *Cheyne-Stokes* breathing is a peculiar rhythmical irregularity of breathing (see § 22). When movement of the chest causes pain, as in pleurisy, or when the muscles of the chest wall are paralysed, there is abdominal breathing. When the diaphragm is out of action, as in certain abdominal conditions, there is exaggerated heaving of the thorax and noisy respiration.

2. *The Shape and Size of the Chest.*—A cross-section of the *healthy* adult chest gives almost the form of an ellipse, the longer diameter being from side to side. In the child it is more circular in shape. The chest should appear symmetrical, although in reality the right side is slightly larger than the left. There should be no marked hollowing anywhere; the clavicle should form only a moderate prominence between the supra- and infraclavicular regions. The circumference of the chest varies with the height of the individual, but it should average for a man 5 feet 6 inches about 34 to 35 inches. With deep inspiration it should expand about 1½ to 2 inches. The measurement at the level of the nipples in the male is a rough measure of the individual chest capacity (see above).

FIG. 33.—CHEST OF EMPHYSEMA. The dotted line represents the normal outline.

The relative shape and capacity of the two sides is measured by a cyrtometer (see also p. 145). The principal abnormalities in shape are the emphysematous, phthisical, and rachitic chests.

The commonest form of abnormality in the shape of the adult chest is the *emphysematous*, or, as it is called, the barrel-shaped chest. Briefly expressed, this alteration consists of the fixation of the chest in a position of permanent inspiration, and expiration cannot be completely performed. The sternum becomes curved, the lower part being unduly drawn in; and a horizontal section shows the chest to be unduly circular (Fig. 33). When the hands are placed flat upon the chest on each side, they readily appreciate the fact that in advanced cases there is elevation, but no lateral expansion of the thorax, during inspiration. The upper ribs are crowded together, whilst the lower ribs are farther apart than normal, and the epigastric angle is very wide. Owing to the permanent elevation of the clavicles and upper part of the chest, and the unusual degree of development of the accessory muscles of inspiration, the neck looks abnormally short in an emphysematous subject.

The *phthisical* chest is too long vertically, and, in section, too rounded.

On inspection from the front it appears flat, but this flatness is more apparent than real, owing to the slipping forward of the scapulæ towards the front of the rounded chest. In the phthisical chest the antero-posterior is larger than the transverse diameter, as in childhood.

The *rachitic* chest is common in children. Owing to the weakness of the bones, the chest acquires a characteristic shape (Fig. 34). A vertical groove occurs at the weakest part of the wall of the chest—*i.e.*, down each side of the sternum, just outside the " rickety rosary " or beaded junction of ribs and cartilages (§ 478). Harrison's sulcus is often present at the same time ; it is a horizontal groove at the level of the xiphoid cartilage, running from the middle line in front obliquely outwards and slightly downwards as far as the mid-axilla, along the costal arch.

The *pigeon-breast* is found in those who have had some obstruction to respiration in early youth, such as that due to adenoids or whooping-cough. The sternum is prominent, the ribs meeting it at a more or less acute angle. The cross-section of the chest is therefore almost triangular (Fig. 35).

Among the irregular or *asymmetrical abnormalities* in the shape of the chest which the student should look for are *hollowing, prominence, or contraction*.

(α) *Localised Hollowing* or " flattening " of the infraclavicular region may indicate phthisis, or any disease rendering the underlying part of lung useless for respiration.

(β) *Undue Prominence* on one side of the ribs anteriorly may be due to : (i.) Scoliosis —*i.e.*, lateral curvature of the spine, the convexity of the chest being in the opposite direction. (ii.) Intrathoracic' tumour, effusion, abscess, or air (pneumothorax) in the chest. (iii.) If the cardiac

Fig 34 —RACHITIC CHEST The dotted line represents the normal outline.
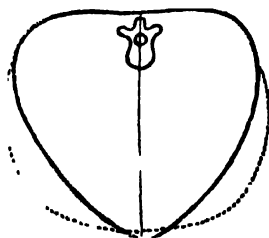
Fig. 35 – PIGEON CHEST. The dotted line represents the normal outline

region be prominent, it may be the result of cardiac disease in early youth, before the ribs were fully developed, and possibly an adherent pericardium. (iv.) An enlarged liver or spleen or abdominal tumour or abscess may also cause a bulging of the lower ribs on the right and left sides respectively. (v.) Subcutaneous emphysema or œdema, a localised deposit of fat or other tumour.

(γ) *Contraction* of an *entire side* of the chest which may be due to : (i.) collapse of a lung (§ 118) ; (ii.) previous empyema (§ 99) ; (iii.) chronic interstitial pneumonia and fibroid phthisis (§§ 114 and 111).

The *Cyrtometer* is an instrument consisting of two flexible pewter or pure tin bands joined by a hinge and graduated in inches. It is used to measure the *relative*

size and *shape* of the two sides. Place the hinge *exactly* opposite the spinous processes posteriorly, and bend the pewter limbs round to the front following the contour of the chest precisely. The instrument is then placed on paper, and the outline thus obtained may be marked by running a pencil round the band.

When possible, X-ray examination is advisable to aid the diagnosis of early tubercle, tumours, and fluid effusions, and for the detection of the position and extent of movement of the diaphragm.

§ 87. **Palpation** is the next step in the routine examination of the lungs. The amount of movement with respiration is estimated better by palpation than by inspection. This test is important in the diagnosis of consolidation at one apex, and in the detection of fluid, tumour, or other cause of deficient activity of one lung or part of a lung. By palpation *Vocal Fremitus* (V.F.), or the vibration of the voice, can be felt. It is scarcely appreciable in women or children with high-pitched voices, but is marked in the adult man. The V.F. is normally greater at the right than at the left apex. This test is of the greatest value in differentiating solid and fluid. Thus the V.F. is *increased* where there is consolidation of the lung, as in pneumonia or phthisis, whereas it is *diminished* or absent when the lung is separated from the chest wall by fluid, thickened pleura, tumour, or air, or when air is not entering the larger bronchi, as in cases of obstruction of a bronchus. Not only is the V.F. a valuable differential sign, but its degree of diminution is a useful measure of the *amount* of fluid present in cases of pleuritic effusion. In bronchitis the rhonchi can be felt, *rhonchial fremitus* ; and in pleurisy and pericarditis *friction* may be distinctly felt by the hand. Tenderness due to broken rib, pointing empyema, subcutaneous emphysema, and external tumours are made out by palpation.

§ 88. **Percussion** is, after palpation, the next step in the examination of the chest. There are two kinds of percussion, *immediate* and *mediate*. In the latter a piece of ivory or wood is placed on the chest, and is struck by a small hammer, or with the finger. The *immediate* is the more usual form of percussion. To elicit the normal resonance of the lungs percussion should be stronger than when applied to make out the cardiac dulness. Begin at the apex and percuss *alternate sides* at exactly corresponding points in order *to compare the healthy and unhealthy sides*, and thus work gradually downwards. Place the first or second finger *firmly* and *flat* against the chest, in a horizontal position—*i.e.*, parallel to the suspected line of dulness. (Only in suspected mediastinal tumour should it be placed vertically.) Then strike upon it with the tips of all the fingers of the right hand. The blow should come from the wrist, not the elbow ; and the " staccato " movement should be imitated. Some use one (the middle), two, or three of the fingers of the percussing hand, but this makes the stroke too light, unless, as sometimes happens, it is desirable to demonstrate the delicate shades of pitch, intensity, and quality of the sound.[1]

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[1] The *pitch* of the note is its position on the scale, and depends upon the frequency of the air vibrations. The *intensity* of a note depends on the amplitude of the vibrating air waves. The *timbre* or quality of a note is a characteristic which depends on the

When examining the *back* of the chest (Fig. 36), the patient should be instructed to cross his arms and bend a little forward so that the scapulæ
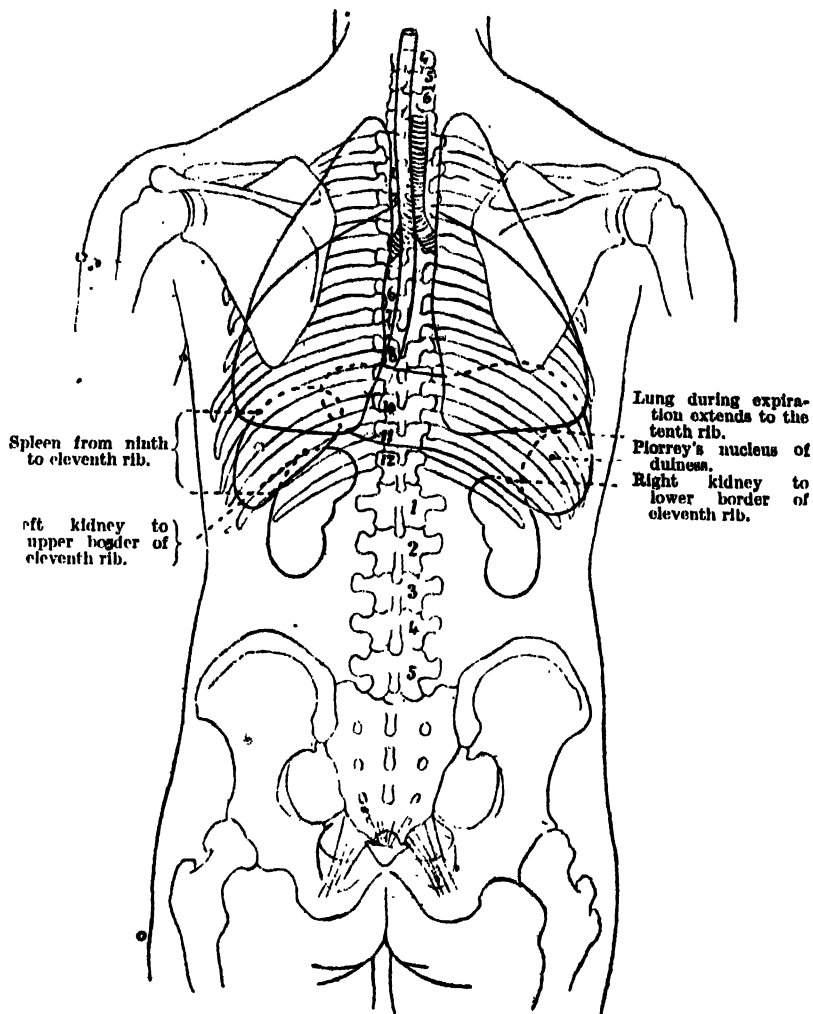

Spleen from ninth to eleventh rib.

Left kidney to upper border of eleventh rib.

Lung during expiration extends to the tenth rib.

Plorrey's nucleus of dulness.

Right kidney to lower border of eleventh rib.

FIG. 36.—THE LUNGS AND OTHER VISCERA FROM THE BACK.—The right lung has three lobes, the left lung two only, and the positions of the great fissures are shown in the figure. The lines on the figure are only approximate guides. When accuracy is desired, the exact position of the lung fissures is obtained by ausculto-percussion. A rough guide to the upper border of the lower lobes is found in the position of the vertebral borders of the scapulæ when the patient crosses his arms in front of him, and places each hand on the opposite shoulder. The great fissure, which separates the middle and lower lobes, on the right side, and the upper and lower lobes on the left side, is indicated on both sides by drawing a line from the second dorsal vertebra to the junction of the sixth costal cartilage with the sternum. The fissure separating the middle and upper lobes on the right side is found by drawing a line from the junction of the fourth costal cartilage with the sternum to meet the line of the great fissure in the mid-axilla.

nature and structure of the vibrating body, just as a wire string and a gut string, though producing the same note, possess a different *timbre* or quality. The *timbre* is largely a question of overtones.

are drawn out of the way. The normal resonance of the lung extends posteriorly to the upper border of the eleventh rib on the right side, and the lower border of the eleventh rib on the left side. On deep inspiration the resonance extends an inch lower, and during deep expiration an inch higher. Owing to the thickness of the scapular muscles the note over the scapulæ may be markedly impaired in muscular people. To examine the *sides* of the chest the patient should be told to put his hands on the top of his head.

The normal pulmonary note can only be learned by practice and experience, and the student should *frequently practise first on normal chests*, so as to accustom himself to the normal resonance; and afterwards on abnormal chests.

The normal percussion note is resonant. It is *dull* or *flat when* the lung tissue is solid, as in pneumonia; or when the chest contains fluid, as in pleuritic effusion, or with a thickened pleura or tumour. The percussion note is *hyper-resonant*, sometimes tympanitic, whenever the lung tissue is unduly open—*i.e.*, too full of air, as in emphysema, when there is a cavity near the surface, or sometimes when there is air in the pleura (pneumothorax). Cracked-pot sound is a modification due to a large cavity (Phthisis, § 110). It is normal in children, in whom it is due to the great elasticity of the ribs. When one part of the lung is over-distended with air, as it is in the part which is above a pleuritic effusion (which compresses the lower part of the lung), or above a pneumonic consolidation, the note is unduly resonant. This kind of resonance is called Skodaic resonance; and it may be almost tympanitic (drum-like) in character. It is due to the relaxation of the healthy lung tissue, and the increased amount of air which it contains.

Gairdner's Line.[1]—It is useful to remember that a line drawn from the left anterior axillary fold to the umbilicus is normally *resonant throughout its entire length*. Abnormally it may be impinged upon anteriorly by consolidation in the upper part of the left lung, cardiac enlargement, or by enlargement of the liver; and posteriorly by consolidation or fluid at the base of the lung, splenic enlargement, or other abdominal tumours.

Increased Resistance is another quality which can be observed in the process of percussion as above described. This property of resistance can perhaps be better elicited by placing a finger of the right hand over an intercostal space and pressing lightly. It is greatest over fluid, and thus affords an important sign in pleural effusion, but is present also in consolidation, though in a less degree. It is a sign which, at first, is difficult to elucidate, but comes to be relied upon, in addition to percussion, by some, especially by those whose appreciation of differences in note is imperfect.

§ 89. Auscultation.—The binaural stethoscope is used by the majority at the present day; its convenience has led to the discarding of the single wooden stethoscope. A stethoscope should always be tested before being purchased. Firm rubber should be employed for the tubing. It is important that the chestpiece be held flat and firmly on the skin of the

[1] It was Sir William Gairdner who emphasised the value of this line in physical diagnosis, but I am not certain if he was the first to do so.

chest. When no stethoscope is available the ear may be placed directly on the chest, with an intervening towel or handkerchief. In auscultation there are four things to be observed : (a) The length and intensity of the respiratory murmur (R.M.) ; (b) the relative length of inspiration and expiration ; (c) the presence of adventitious sounds within or outside the lungs ; and (d) the voice-sounds or vocal resonance (V.R.).

(a) The normal CHARACTER OF THE BREATH SOUNDS—i.e., the vesicular or " respiratory murmur " (R.M.), caused by the air entering and leaving the air vesicles, should be listened to in healthy chests as often as possible. It has a soft whiffing character ; expiration can hardly be heard, but if heard there is normally no pause between it and inspiration. The R.M. is normally very loud in children, and when a loud R.M. is met with in adults, it is called " puerile breathing." The breath sounds audible over the right apex are more pronounced than on the left side ; the difference varies with the age and the build of the patient. The breath sounds are INCREASED—i.e., the breathing is " tubular " or " bronchial " [1] when the lung is solid, as by tubercle, pneumonia, or collapse, or when a new growth lies between the larger bronchial tubes and the surface. In this condition the sound produced in the glottis is conveyed down the bronchi direct to the ear, owing to the increased conductivity of the solid lung substance. Bronchial breathing can be heard normally by listening over the upper segment of the sternum, or near the fourth dorsal vertebra at the back. It has three features—inspiration and expiration are of equal length and character, have an interval between them, and are both rough. Cavernous respiration is exaggerated tubular breathing, and is heard when the sound produced in a dilated bronchus or cavity is conveyed in like manner to the surface. Cavernous respiration is normally heard over the trachea. Amphoric breathing is a sound like air entering a bell-jar, and is sometimes heard over pneumothorax or a very large cavity. The breath sounds (R.M.) are DIMINISHED or absent when a layer of fluid, tumour, or a thickened pleura intervenes between the lung and the chest wall, or when the air does not enter the lung tissue owing to obstruction in a bronchial tube.

(b) The RELATIVE LENGTH OF INSPIRATION AND EXPIRATION is approximately as 10 to 12, but heard through the stethoscope, the inspiratory is three times as long as the expiratory sound, which follows it without a pause. Expiration is prolonged in any disease which involves a loss of elasticity of the lung tissue, such as emphysema, or tubercle in an early stage.

(c) The presence or absence of ADVENTITIOUS SOUNDS has next to be noted. (i.) Pleuritic friction is produced by the two inflamed and roughened surfaces of the pleura rubbing together. (ii.) Within the lung various moist sounds may be added to the respiratory murmur. Thus the presence of excessive mucus or other fluid in the large bronchial tubes gives rise to

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[1] The terms " bronchial " and " tubular " are generally taught as synonymous, but some schools teach that there are three kinds of bronchial breathing—high-pitched, or tubular ; medium-pitched, or true bronchial breathing ; and low-pitched, or cavernous breathing.

"large or bubbling râles" as the air bubbles through the fluid. When the small tubes or air cells are similarly affected, "small mucous râles" or "crepitations" are heard which resemble the rustling of tissue-paper or hairs rubbed together near the ear. They are audible in cases of early pneumonia and œdema of the lungs. When these adventitious sounds are few and difficult to detect, they become clearer when the patient draws a deep inspiration immediately after a slight cough. Rhonchi are continuous sounds, supposed to be produced by the air passing over the turgid and swollen mucous membrane of the bronchi. When low-pitched, they are *sonorous*, resembling the snoring of a sleeper; when high-pitched, they are "sibilant" or "whistling." Rhonchi are often hard to distinguish from friction sounds, but it may be remembered that, whereas friction sounds heard during inspiration and expiration are separated by a short but distinct interval of silence, rhonchi are not so separated, but fade one into the other. Crepitations sometimes resemble friction sounds, but are distinguished by being audible only during inspiration.

(*d*) The VOICE SOUNDS, or vocal resonance (V.R.). (i.) When the patient speaks, the vocal resonance is INCREASED (*bronchophony*) over a cavity, or if the conductivity of the lung substance is rendered greater by consolidation, such as that produced by tubercle or pneumonia. If this be so great that even whispered words are conducted, it is known as *whispering pectoriloquy*. Some authors confine this term to the very exaggerated whispering sounds which are heard over large smooth-walled cavities, and consider that the term should be used only in those cases where the spoken or whispered voice is particularly clear and distinct in addition to being loud and close. (ii.) The vocal resonance is DIMINISHED when a layer of fluid or air intervenes between the lung and the chest wall (*e.g.*, in pleuritic effusion and pneumothorax), or when there is a thickened pleura. Nevertheless, in a slight pleuritic effusion and at the upper level of a moderate effusion the higher tones of the voice sounds are sometimes conducted, especially at the angle of the scapula, and resemble the bleating of a goat (hence called *Ægophony*).

Clinically, all the diseases of the lungs may be conveniently divided into those with **dulness on percussion**, those in which the percussion note is **normal**, and those in which it is **hyper-resonant**. Those with **dulness** may be subdivided into two groups— those in which the dulness is due to CONSOLIDATION, and those in which it is due to FLUID. The clinical features by which solidification of the lung is distinguished from fluid in the chest are so important that they are given in a tabular form.

	Consolidation of Lung.	Pleural Effusion.
INSPECTION.	Movement impaired. .. .. May be flattening over the part (if infraclavicular region).	Movement impaired. May be bulging (of intercostal spaces).
PALPATION.	V.F. INCREASED.	V.F. DIMINISHED or absent.
PERCUSSION.	Resonance impaired.	Absolutely dull over fluid.
AUSCULTATION.	BREATHING TUBULAR. .. V.R. INCREASED.	B.M. ABSENT OR WEAK. V.R. DIMINISHED.

Ausculto-Percussion, when employed by experienced observers, enables them to define the boundaries of the heart, or of a mediastinal tumour, with greater accuracy. It is useful to determine the lobe in which disease is situated. In this method the stethoscope (preferably a binaural) is placed over the middle of a lobe, while one coin is tapped on another, first over another lobe, and then over the same lobe as that to which the stethoscope is applied. The listening ear recognises the difference of the impact in the two cases. The coins are then placed over the supposed margins of the lobes, and by the slighter or stronger impact conveyed to the ear the division between the lobes can be readily defined. In pneumothorax the pathognomonic " bell-sound " is obtained by this method.

§ 90. Fallacies in Diagnosis of Diseases of the Chest.—This list includes the most important fallacies, but it is impossible to make it exhaustive.

1. When the chest wall is very thin the sounds heard on auscultation are proportionately loud. The percussion note is also louder, and it is consequently easy to fall into the error of supposing the emphysema is present. In children the breath sounds are always more distinct than in adults, and are, moreover, more readily conducted, so that adventitious sounds having their origin on one side may even be heard quite plainly on the other.

2. A chest wall with excess of subcutaneous fat or œdema will give rise to error if it be not borne in mind that the sounds on auscultation and percussion are alike deadened and indistinct. The sounds heard over the scapular region are always less distinct than those heard elsewhere. When a patient does not breathe deeply, owing to debility or pain on movement of the chest, or when the chest wall is very fat, the breath sounds may be almost inaudible.

3. The presence of much hair on the chest, as it is rubbed by the stethoscope, gives rise to sounds like fine crepitations.

4. The sounds created by friction of the shoulder joint often lead to mistaken diagnosis of pleurisy at the apex.

5. The fault of applying the stethoscope to the ear, instead of the ear to the stethoscope, often leads to the chest piece being only in partial apposition to the chest, an error which causes misleading sounds to reach the ear. The friction between the rubbers of the stethoscope may originate sounds which are misinterpreted.

6. It is well to remember that dulness on percussion does not necessarily mean that there is fluid or consolidation present. It may also be caused by thickened pleura and by the presence of tumours. The latter may be outside the chest, but pushing up into the thorax—e.g., hepatic or splenic enlargement, subdiaphragmatic abscess.

7. Tumours of the chest wall will sometimes lead to the impression that there is some difference in the size of the two sides of the thorax, and this difference may be referred to some morbid condition of the chest contents. The swelling caused by subcutaneous emphysema or bloodclot, both of which may follow an accident, gives rise to a faint crepitation which may be easily mistaken for the signs of injury to the lung beneath.

8. When one lung has been long out of action, as in fibroid phthisis, the other undergoes compensatory enlargement and encroaches on the affected side of the chest. The hypertrophied lung gives rise to sounds identical with those of emphysema.

9. The breath sounds are better heard and the percussion note is higher at the right than at the left apex, owing to the presence of the eparterial bronchus on the right side.

10. Atrophy of the muscular tissues about one shoulder leads to an apparent flattening on that side very like that seen in phthisis.

11. Peritoneal friction, due to inflammation below the diaphragm, may be mistaken for pleuritic friction, as it is frequently audible at the base of the lungs, and as far up as the seventh interspace.

12. Distension of the abdominal organs, as in meteorism, may extend high up into the chest and simulate hyper-resonance of the lungs. This is especially probable

when the lungs have been drawn up with adhesions or fibroid contraction. A hernia of the diaphragm with protrusion of the stomach, or the opening of an abdominal abscess into the chest, may cause amphoric echoes and bell sounds, as in pneumothorax.

13. Dextro-cardia is very rare, but it is necessary to be on one's guard lest it be rashly supposed that the heart is displaced by effusion or by some tumour.

14. Finally, it is well to remember that the presence of lung signs usually found in association with acute disease must always be interpreted with due regard to the constitutional condition and co-existing signs of disease in other organs.

§ 91. Examination of the Sputum.—Much may be learned from an examination of the sputum. First, as regards its Appearance. In simple pleurisy, though the cough is distressing, expectoration is absent (*i.e.*, the cough is " dry "). If the disease be confined to a moderate catarrhal process of the bronchial tubes (*e.g.*, bronchitis), the sputum is white, clear, and frothy (" mucous expectoration "). If the process be more severe and suppurative, or if the lung tissue be breaking down, then pus is present, and the sputum is yellowish (muco-purulent). This watery sputum is expectorated in large quantity in œdema of the lungs. In phthisis, when the lung is breaking down, the sputum is often voided in thick purulent masses like coins, hence called *nummular*. In cases of pulmonary abscesses, tuberculous cavities, and of empyema bursting into the lung, large quantities of almost *pure pus* are expectorated from time to time. Extremely fœtid expectoration is voided in gangrene of the lungs and in bronchiectasis. The latter is distinguished by having large quantities of *putrid sputum*, brought up by paroxysms of violent cough at *one time;* while in the intervals the cough and expectoration are those of bronchitis. The bronchiectatic sputum, on standing, separates into three layers—the upper clear and frothy ; the middle granular, with mucus ; the lower purulent, with thick " Dittrich's plugs " (p. 153). The foul odour is due to valerianic and butyric acids. In *pneumonia* the sputum is very characteristic, being (i.) almost airless and extremely viscid, so that the vessel containing it may be inverted without spilling it, and (ii.) tinged with blood, thus having a " rusty " colour. In severe cases, and in new growth of the lung, the sputum becomes thinner, frothy, and dark red, the " prune-juice " sputum. *Casts* of the bronchial tubes, which can be seen by the naked eye (Fig. 45, p. 176), are expectorated in plastic bronchitis, and occasionally in croupous pneumonia, and shreds of membrane in diphtheria. Hydatid cysts, resembling empty gooseberry-skins, are expectorated in that rare condition hydatid disease of the lungs, or when hydatid of the liver ruptures into them. In town dwellers, and those with dusty occupations, the sputum is dark, or even black, from the presence of carbonaceous and other particles. " Anchovy sauce " coloured sputum is characteristic of abscess of the liver which has burst into the lung (§ 269).

FIG. 37.—Elastic Fibres.

FIG. 38.—Charcot-Leyden Crystals in Sputum.

Microscopic Examination of the Sputum.—Various *bacteria* and *fungi* (*e.g.*, tubercle, pneumococcus, influenza, pyogenic cocci, anthrax, glanders, plague, the fungi of actinomycosis, blastomycosis, and aspergillosis) may be found in the sputum. The method of detecting these is described in Chapter XX.

In all destructive diseases of the lung fragments of pulmonary tissue are present —i.e., epithelial cells and connective tissue. The most characteristic is *elastic tissue*. Elastic fibres are best revealed by taking a small portion of the sputum and boiling it with liquor potassæ, which breaks up and renders clear all the other elements, but leaves the elastic fibres unattached. These sink to the bottom of the test-tube, and

may be withdrawn by a pipette (precautions, see Urinary Deposits) for examination under the microscope. They appear as wavy, highly refractile fibres, of uniform thickness, with square-cut ends, and are typically arranged as if surrounding an air cell (Fig. 37). Elastic tissue is found in the mouth after meals, so the mouth and teeth should, as a precaution, be cleansed before the observation is made ; but circularly arranged elastic fibres are quite distinctive of breaking-down lung tissue. The " Dittrich plugs " of a bronchiectatic sputum (§ 121) are little pellets, which contain pus and epithelial cells, with needle-shaped fatty acid crystals. They are believed to be pathognomonic of bronchiectasis. Sometimes elastic fibres are also present in small amount.

Curschmann's spirals are found in the sputum of asthmatic patients. They form pellets or *perles*, the size of sago grains, which can be uncoiled to form a thread about an inch long. Microscopically, they are seen to consist of fine mucous fibrils wound spirally round a central core of mucus. They are probably allied to small bronchial casts (Finlayson). *Charcot-Leyden* crystals (Fig. 38) are colourless, pointed, octahedral crystals, formerly supposed to be pathognomonic of asthma, but now known to occur in the sputum of plastic bronchitis also. They have also been found in the blood of leukæmia. *Hæmatoidin* crystals are brown or yellow needles or plates, found in cases of old hæmorrhage from any cause. *Cholesterin, leucin,* and *tyrosin* crystals are found occasionally in cases where the sputum has been purulent for a long time. Various *parasites* (streptothrix, echinococcus, Distoma pulmonale, etc.) are sometimes found in the sputum. Sarcinæ and Oïdium albicans come usually from the alimentary tract.

PART C. DISEASES OF THE LUNGS AND PLEURÆ : THEIR DIAGNOSIS, PROGNOSIS, AND TREATMENT

§ 92. Classification.—For practical purposes, diseases of the lungs and pleuræ may be divided into ACUTE and CHRONIC, and each of these may be subdivided into those without dulness, those with dulness, and those with hyper-resonance.

	Acute.	Chronic.
WITHOUT DULNESS	I. Acute Bronchitis. II. Dry Pleurisy. III. Acute Phthisis. IV. Whooping-cough. V. Acute Pulmonary Œdema.	I. Chronic Bronchitis (and Plastic Bronchitis).
WITH DULNESS	Common. { I. Pleurisy with effusion (and Empyema). Rare. { II. Lobar Pneumonia. III. Lobular Pneumonia.	I. Chronic Phthisis [1] (and Fibroid Phthisis). II. Hydrothorax. III. Pulmonary Congestion (or Œdema). IV. Interstitial Pneumonia. V. Thickened Pleura. VI. Cancer and other neoplasms. VII. Collapse of the lung. VIII. Syphilitic disease.
HYPER-RESONANCE	I. Pneumothorax.[2]	I. Emphysema.

[1] There is no dulness in quite the early stages of some cases.
[2] Pneumothorax is often part of a chronic disease, when the onset is hardly noticed.

Paroxysmal.	Diseases recognised by the Character of the Sputa.
I. Asthma.	I. Bronchiectasis.
II. Acute Pulmonary Œdema (sometimes).	II. Gangrene of the lung.
	III. Abscess of the lung.
	IV. Actinomycosis and other diseases due to fungi.

§ 93. The **Routine Procedure** here resembles in principle that of diseases of the heart. First, *What is the patient's leading symptom?* If suffering from lung disease, his cardinal symptom will be one of those mentioned in section A. Breathlessness and cough are the chief cardinal symptoms.

Secondly, follow this up with a few questions to ascertain the *history of his illness*, and especially whether *the disease be acute or chronic*. Other important points are whether the patient has been exposed to a " chill," and whether there is any " lung disease " in the family. Do not use the word " consumption " ; it may frighten your patient unnecessarily.

Thirdly, proceed to the PHYSICAL EXAMINATION OF THE LUNGS. The routine method is as follows :

1. Ascertain whether there is any increased rate or other modification in the breathing or alteration in the shape of the chest (by *inspection*, and, if necessary, by measurement).

2. Ascertain if there be any dulness or hyper-resonance (by *percussion*).

3. Listen to the breath and voice sounds, directing special attention to any part suspected of disease (by *auscultation*).

4. Test the vocal fremitus by *palpation*.

5. The sputum should be inspected, and, if necessary, examined microscopically.

The chest should always be stripped, and it is more convenient to examine the patient in a sitting posture, if he be not too ill.

If the illness developed gradually, and is of some standing, and unattended by marked constitutional disturbance, then turn to **Chronic Pulmonary Disorders** (§ 106, p. 173).

If the illness came on recently and suddenly, accompanied by fever, quickened respiration, coated tongue, and with marked malaise, then the case is one of the **Acute Pulmonary Diseases**, below.

There is one disease of the lungs, ASTHMA, which comes on in sudden acute attacks from time to time ; it is chronic, with acute exacerbations (§ 105, p. 172).

Acute Diseases.—We now proceed to percuss the chest. In all acute diseases special attention should be directed to the lower and back part of the chest just below the scapulæ. Careful percussion of this region will give us important aid in diagnosis.

TABLE VI.--DIAGNOSIS OF COMMON ACUTE DISEASES OF THE
LUNGS AND PLEURÆ.

	Percussion Note.	Auscultation
Acute Bronchitis . . .	Normal	R.M. and V.R. normal; Loud moist râles and dry rhonchi.
Dry Pleurisy	Normal	Breath and voice sounds normal; Pleuritic friction.
Acute Pulmonary-Tuberculosis	Normal, or scattered areas of dulness	Scattered fine moist râles may be the only auscultatory signs.
Pleurisy with effusion .	Dull	R.M., V.R. and V.F. diminished; Pleuritic friction at early and late stage.
Lobar Pneumonia. . .	Dull	V R. and V.F. increased · Bronchial breathing; Fine or coarse (redux) crepitations.
Lobular Pneumonia . .	Scattered areas of dulness	Fine crepitations and scattered areas of bronchial breathing with increased R M and V.R. obscured by rhonchi and râles.

The acute diseases without alteration in the percussion note, *i.e.*, **without dulness**, excluding WHOOPING-COUGH, which is an infective disorder and has no physical signs in the lungs peculiar to it, and ASTHMA, which is of a paroxysmal character—are : ACUTE BRONCHITIS ; DRY PLEURISY ; one form of ACUTE PULMONARY TUBERCULOSIS ; [1] and ACUTE PULMONARY ŒDEMA.

I. *The patient complains of a cough, with frothy expectoration, and his temperature is slightly elevated ; there is no **alteration** in the percussion note but on auscultating the chest, loud* RHONCHI *are heard.* *The disease is* ACUTE BRONCHITIS.

§ 94. **Acute Bronchitis,** or inflammation of the bronchial tubes, is certainly the most common acute disease of the lungs in this climate.

Symptoms.—The disease commences gradually in the course of one or two days, with a feeling of tightness of the chest, of soreness behind the sternum, shortness of breath, frequent cough, and slight rise of temperature, 100° to 101° F. The inflammatory process lasts from ten days to three weeks, and gradually subsides. The sputum is viscid and scanty during the first few days, and then becomes thinner, muco-purulent, and more easily coughed up.

Physical Signs.—The percussion note is unaltered unless, as so frequently happens, emphysema be present also, in which case the chest is unduly resonant. On auscultation the vesicular murmur is obscured over the whole chest on both sides by loud rhonchi and moist râles (see Fig. 39) which are variable and altered by coughing. On palpation rhonchial fremitus can frequently be felt.

Causes.—Bronchitis is generally attributed to : (i.) A chill ; that is to say, sudden exposure to cold, with a "determination of blood to the interior."

[1] In the early phase of this malady there is no alteration of the percussion note, but as the disease progresses a patchy dulness appears, if the patient live long enough.

(ii.) Sometimes, however, it is caused by spreading from laryngitis. (iii.) It is a frequent complication of many of the specific fevers, especially measles, whooping-cough, and typhoid. It is so frequently present with the first and last as to constitute an aid to the diagnosis of those diseases. (iv.) Certain occupations which expose people to irritating vapours and small particles of dust predispose to acute bronchitis. Thus the cotton-mill hands and chemical manufacturers frequently suffer from bronchitis.
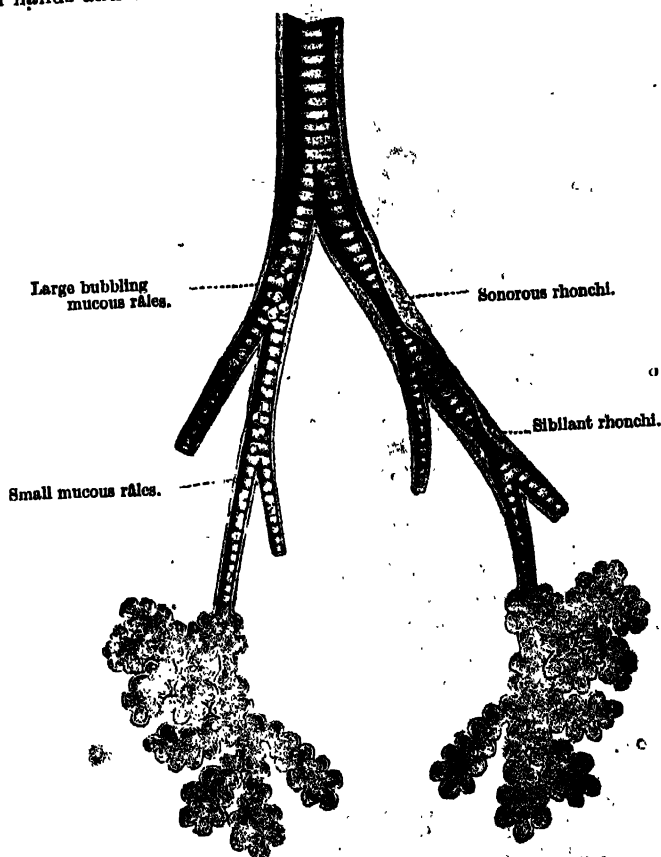

Large bubbling mucous râles.

Sonorous rhonchi.

Sibilant rhonchi.

Small mucous râles.

FIG. 30.—Diagram to show the production of râles (moist sounds) on left, by mucus in bronchial tubes, and rhonchi (dry sounds) on right, by narrowing of the tubes.

It is also common amongst cabmen, mariners, and others who are exposed to all weathers. (v.) It is a common accompaniment of many other pulmonary diseases, though it may be a subordinate feature ; and (vi.) it is commonest in childhood and old age.

The *Diagnosis* is not difficult in most cases, but *acute tuberculosis* is at first very apt to be regarded as acute bronchitis. The diagnosis is aided by the greater elevation, and the intermitting character of the pyrexia

in the former, and by the presence of the tubercle bacillus in the sputum. The " *capillary bronchitis* " of children is really a *broncho-pneumonia* (*q.v.*) ; the constitutional symptoms and dyspnœa are much more marked, there may or may not be some dulness, and the differentiation from simple acute bronchitis is not always easy.

The *Prognosis* is favourable in adolescence and adult life, and it always clears up in one to three or four weeks, though it has a special liability to return, and ultimately to become chronic. It is dangerous in infancy and old age, where the resisting powers are feeble. It is one of the commonest causes of death in the latter. If an attack of acute bronchitis does not begin to clear up in two or three weeks, pulmonary tuberculosis should be suspected, especially if the patient be young.

Treatment.—The indications are : (i.) During the first stage, to promote the secretion ; (ii.) when the secretion is free, to stimulate the bronchial mucous membrane ; (iii.) during convalescence, to improve the general condition so as to enable the patient to throw off his liability to bronchitic attacks. At the onset give an aperient and a diaphoretic mixture, with perhaps a few grains of Dover's powder to soothe the pain. Poulticing is useful, and emetics are given to children. To promote the flow of secretion warm alkaline drinks and expectorants such as ipecacuanha and antimony, together with liq. ammon. acet., are especially useful. When the secretion is free—that is, after three or four days—stop the antimony, and administer expectorants, such as ammonium carbonate, syrup of tolu, senega, and squills (Formula 57). If the patient is of a gouty or rheumatic diathesis, or the sputum is very tenacious, add potassium iodide to the expectorant mixtures. The patient must be confined to bed, and will derive great benefit from the inhalation of steam. In childhood, this is best done by a bed canopy and a steam kettle beside it ; in adults, a kettle with a long spout on the fire will suffice. Linseed-meal poultices, a turpentine stupe to the chest, or a covering of cotton wool give great relief to the distressing tightness of the chest (see also Formulæ 30 and 68). During the stage of recovery tonics and cod-liver oil are called for.

Two varieties of bronchitis were frequent amongst the troops. (i.) *Purulent bronchitis* attacks healthy young adults, and is accompanied by extreme distress, cardiac embarrassment, rapid pulse, cyanosis, non-aërated sputum, and patchy areas of collapse. The temperature resembles that of pneumonia and falls by crisis or lysis. (ii.) A variety of acute pulmonary œdema associated with *Capillary bronchitis*, and characterised by large quantities of frothy liquid sputum. Bleeding and belladonna gave the best results, but the disease was often fatal.

A rare form due to a nematode worm is met with in the East (Distomiasis, § 441).

II. *The patient complains of sharp* PAIN *in the chest on inspiration ; he has a short dry cough, and his temperature is moderately elevated ; on auscultation,* FRICTION *sounds are heard. The disease is* DRY PLEURISY.

§ 95. Dry Pleurisy is inflammation of the pleura without effusion. In this disease there is a fibrinous exudation on the visceral and parietal

layers of the pleura, and a tendency to the formation of adhesions, and to the effusion of fluid.

Symptoms.—The disease in some cases comes on quite suddenly with a stitch-like pain in the chest. The constitutional disturbance is never very great, and the patient rarely takes to his bed. The temperature may rise to 100° or 101° F., rarely higher. The most obvious symptom in this disease is pain in the chest, affecting one side only in most cases, and characterised by being greatly increased on deep inspiration. The pain is caused by the contact of the inflamed pleural surfaces, and is usually, though not necessarily, located over the diseased part.

Physical Signs.—Percussion reveals nothing. On auscultation, the respiratory murmur may be found to be normal or shortened, as the patient endeavours to restrain the movements of the chest on account of the pain so caused. From the very outset a pleuritic rub is heard over one side, often most marked at the angle of the scapula (compare § 89). Sometimes the inflammation undergoes resolution or adhesion, sometimes it goes on to effusion. As effusion takes place, the pain and pleuritic friction disappear, to reappear again when this subsides.

Causes.—(i.) Sometimes it is a primary malady, attributed to chill, especially in persons of a gouty or rheumatic diathesis. (ii.) It may occur as a complication of some acute infective disease, such as measles or scarlatina. (iii.) Inflammation may extend from disease of the under-lying lung, such as pneumonia, tuberculosis, cancer, and embolism, or from adjacent organs, such as the liver or spleen. (iv.) Undoubtedly a large number of apparently simple pleurisies are tuberculous in origin (some go so far as to say 82 per cent.) ; this fact should always be remembered.

The *Diagnosis* from *muscular rheumatism* (pleurodynia) is made by the tenderness and absence of friction sound in the latter. In *intercostal neuralgia* there are tender points along the course of the nerve, and the pain is not aggravated by deep inspiration. Pleuritic friction is distinguished from the rhonchi heard in *bronchitis* by there being in nearly every case of pleurisy a distinct interval between the inspiratory and the expiratory rub.

Prognosis.—It is not a serious malady, and readily yields to treatment ; but sometimes effusion occurs (Pleuritic Effusion, § 98). When this effusion becomes purulent (§ 99) the prognosis is graver. Thickening of the pleura may result, especially in tuberculous cases.

Treatment.—Considerable relief is derived by simply strapping the affected side of the chest, so as to limit the costal movements of respiration. This may be combined with some local application ; that which gives greatest relief is undoubtedly a linseed-meal poultice. As the disease becomes chronic, counter-irritants are called for, more especially iodine, which may be painted on daily until the skin becomes sore. If it does not disappear in the course of a few weeks, we must suspect some other cause for the mischief, such as those mentioned under pleurisy with effu-

sion. Diuretics, diaphoretics, iron, and other tonics are useful. Breathing exercises may be of service during convalescence to prevent the formation of adhesions and correct any tendency to collapse of the lung.

III. *The patient exhibits the signs of subacute bronchitis ; but he has* SEVERE MALAISE *and a* HECTIC TEMPERATURE, *and the sputum may contain* TUBERCLE BACILLI. *The disease is* ACUTE PULMONARY TUBERCULOSIS.

§ 96. **Acute Pulmonary Tuberculosis** (acute phthisis, galloping consumption) is a catarrhal process affecting the entire lung tissue, due to the invasion of the tubercle bacillus. It is often part of a tuberculous process infecting the whole body, and is therefore sometimes described as the pulmonary form of acute general tuberculosis (see Chapter XV, where a chart is given showing the typical course of the temperature in both diseases).

Symptoms.—The malady is of most insidious onset, with progressive weakness and emaciation. Some weeks before any physical signs are evident the thermometer shows the typical intermittent pyrexia so characteristic of tubercle—an evening elevation of 101° to 103° F., and a morning normal temperature. In rare cases the inverse type is present, when the temperature is higher in the morning than in the evening. Night-sweats and cough are present, with muco-purulent expectoration. Dyspnœa, and sometimes cyanosis, develop out of proportion to the physical signs, the latter symptom may be extreme, and of itself is a very characteristic feature. Great weakness ensues, and in the third or fourth week the patient may develop the symptoms of the typhoid state.

The *Physical Signs* referable to the lungs are indefinite, or resemble at first those of bronchitis. At first there is no alteration in the percussion note, but by and by careful percussion discovers scattered patches of impaired resonance. Auscultation at first may give little help, but in the course of a week or so it reveals rhonchi and fine râles over certain areas, which do not shift from place to place, as in bronchitis. Later on the râles are coarse and bubbling, and areas of tubular breathing may be found.

The *Diagnosis* in the first stage from bronchitis and broncho-pneumonia is extremely difficult. We have to rely upon the disproportionate emaciation and cyanosis, the character of the temperature, and the patchy distribution of the physical signs in tuberculosis. In other cases the malady is almost indistinguishable from enteric fever except for the marked predominance of the pulmonary signs and the absence of the roseola, and the Widal test is negative. In all stages the detection of the tubercle bacillus in the sputum is a valuable aid to diagnosis, though its absence does not exclude acute pulmonary tuberculosis.

Causes.—The disease may occur at any age, but is commonest in young adults, and in those with a family history of consumption. In some instances acute general tuberculosis originates from a primary focus, such as a tuberculous joint, which had been considered cured. Sometimes the disease follows measles or whooping-cough in children.

Prognosis.—The disease is almost uniformly fatal in about two to twelve weeks. *Treatment* is almost entirely symptomatic.

IV. *The patient, a child, has* PAROXYSMS *of coughing which terminate in a* WHOOP, *and frequently in* VOMITING ; *there is very slight feverishness, but the only signs in the lungs are those of a little bronchial catarrh. The disease is* WHOOPING-COUGH.

Whooping-cough (Pertussis) is an acute infectious disease, and it is described among the microbic disorders (§ 395).

V. *The patient is suddenly seized with acute dyspnœa and copious frothy sputum flows from the mouth and nose. The disease is* ACUTE PULMONARY ŒDEMA.

§ 97. Acute Pulmonary Œdema. *Symptoms.* The sudden onset of acute dyspnœa, with copious, often blood-stained (rose-coloured) sputum, are most characteristic. The diagnostic point about the sputum is that it contains albumen. The face is pale, the expression is one of intense anxiety; there may be a cold sweat. The pulse is feeble, and there may be pain or a feeling of oppression in the chest. The disease is conjectured to depend on weakness of the left ventricle, allowing the accumulation of fluid in the lungs. It may arise in the course of heart disease, more especially aortic disease, arterio-sclerosis, pregnancy, epilepsy, angio-neurotic œdema, acute infections, or Bright's disease. The *physical signs* consist of râles and crepitations which are heard all over the chest.

Treatment.—Sometimes the disease is so rapidly fatal that no treatment is of avail. The best emergency treatment is blood-letting to 20 ounces. In fulminating cases this should be undertaken without delay. Atropine and belladonna have an almost specific action; $\frac{1}{100}$ gr. (0·006) of the former should be given hypodermically at the earliest possible opportunity. The recurrence of attacks cannot be prevented except in those cases where the patient is able to foretell their coming. In these a dose of atropine in time will ward off or very much mitigate the attack. The only prophylactic treatment is directed to the presumed cause of the attacks—*i.e.*, to the underlying disease. The disease may never recur, but in some patients may persist at variable intervals for years.[1]

We now turn to the **Acute Diseases with Dulness on Percussion**— I. Pleurisy with Effusion (Serous or Purulent) ; II. Pneumonia and III. Broncho-pneumonia.

I. *The patient has a* DRY COUGH, *with moderate fever and other constitutional symptoms. On examining the chest, the respiratory murmur, vocal resonance, and vocal Tremitus are found to be diminished or absent. The disease is* PLEURISY WITH EFFUSION:

§ 98. Acute Pleurisy with Effusion.—When describing acute Dry Pleurisy (§ 95) it was pointed out that the disease may undergo resolution or result in adhesions. It may also go on to effusion—Pleurisy with Effusion.

Symptoms.—There is usually a history of a more or less acute onset with pain in the side (§ 95), but as the disease progresses, and the surfaces of the pleura are separated by fluid, pain becomes less and less marked. The patient suffers from general malaise, and finds it difficult to lie on the sound side, because the action of the healthy lung is thereby impeded. A degree of breathlessness may be present, but even with a large amount of fluid this is not invariably a prominent feature.

Physical Signs (see Fig. 40).—Percussion reveals absolute dulness over the fluid. Above the level of the fluid, if the lung be otherwise healthy, there is a hyper-resonant note (Skodaic resonance). When the effusion is large it causes displacement of organs, which may be very considerable (see Fig. 41). The level of the fluid shifts with the position of the patient, especially when much of it is present. On auscultation over the fluid, the breath sounds are absent ; the vocal resonance is greatly impaired or lost.[2] At the upper margin of the fluid posteriorly—perhaps just about

[1] Leonard Williams, the *Lancet*, December 7, 1907, and discussion in subsequent numbers.

[2] Bronchial or tubular breathing occurs wherever there is moderate effusion, because the lung collapses with the progress of the effusion.

the angle of the scapula—only the highest pitched tones of the voice are transmitted, and they produce, therefore, a sound like the bleating of a goat (ægophony). On palpation, the vocal fremitus is found to be diminished or absent over the fluid, and there may be bulging of the intercostal spaces. The amount of fluid present may be estimated by (i.) the degree of diminution of the vocal resonance and fremitus, and (ii.) the amount of displacement of organs. The diagnosis of pleurisy in its earlier stages is referred to under Dry Pleurisy. The differentiation of the physical signs of fluid in the chest, as compared with those of consolidation of the lung, is so important that it is given in a tabular form in § 89. It is sometimes difficult to make out the left margin of the cardiac area when there is effusion in the left pleura. Dr. S. H. Habershon has suggested a very valuable aid in such cases. Place a vibrating tuning-fork, such as aural surgeons use, in mid-axilla over the seventh rib. Listen with the stethoscope over the centre of the cardiac area, and gradually move it towards the tuning-fork, and in other directions. As the stethoscope crosses the boundary of the heart, there is a distinct difference in the note heard through the stethoscope, and in this way the cardiac boundary may be determined. Hydatid of

- - Skodaic resonance.

Friction sound often heard best near angle of scapula.

Percussion note dull; absence of respiratory murmur; absence of V.F. and V.R.

FIG. 40.—Diagram to show physical signs produced in different positions in ACUTE PLEURISY WITH EFFUSION.

the liver may be mistaken for pleuritic effusion, but the dulness has a more rounded outline. X-ray examination shows clearly the position of the fluid.

Course and Prognosis.—In about a fortnight from the date of onset the fluid usually shows signs of diminution in quantity, the vocal fremitus and resonance return, and the breath sounds become more audible. This is the usual course, but several untoward results may ensue : (i.) The effusion may remain for an indefinite time, and re-collect after tapping. (ii.) Adhesions may take place between the two layers, and considerable thickening of the pleura result. (iii.) The fluid—especially in children after scarlatina—may become purulent (Empyema, see below).

C.M.   M

Treatment.—To get rid of the effusion purgatives, diuretics, and dia-phoretics (potassium citrate and bitartrate, potassium nitrate, liquor ammoniæ acetatis, etc., Formula 55) are often efficacious. Counter-irri-tants may be useful. Iron and other tonics are useful. Autoserotherapy has had success abroad in pleural and peritoneal effusions. One to ten c.c. of the serous fluid are aspirated; the needle is withdrawn as far as the sub-cutaneous tissue, where its contents are injected. If these measures fail after a few weeks' trial, paracentesis should be performed (Figs. 41 and 42). Under certain conditions it is inadvisable to delay paracentesis: (i.) A large effusion (*e.g.*, with dulness extending upward as far as the third rib; (ii.) cardiac embarrassment, as evidenced by cyanosis, palpitation, and a rapid pulse; (iii.) respiratory embarrassment, shown by urgent dys-pnœa and paroxysmal attacks of coughing; (iv.) effusion in the other pleura, or œdema of the other lung. It should be remembered, in recur-rent effusion, that tubercle may be the cause.

FIG. 41.—Case of PLEURITIC EFFUSION, showing displacement of organs (heart and liver). The patient was a boy aged twelve, admitted under the care of Sir William Gairdner in the Western Infirmary, Glasgow, April 20, 1895.
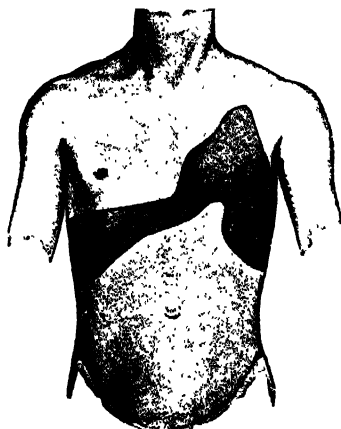
FIG. 42.—Shows altered state of dulness under use of diuretics (April 29). Lin. hydrarg. was applied, and he took in-ternally Pot. cit. gr. x., Pot. iod. gr. v., Liq. am. acet. ℥ xx., Decoct. scopar. ℥ii., quartis horis. Patient also had diarrhœa at this time.

Paracentesis Thoracis.—The instrument used is an adaptation of the familiar troçar and cannula. We are here dealing with a cavity whose contents are under a minus pressure, so it is necessary to have a pump or exhausted bottle communicating with the troçar. The site of puncture—usually the seventh interspace in the posterior axillary line—should be cleansed with ace-tone or other strong antiseptic. The needle of the aspirator must be aseptic. The bottle or chamber of the syringe is next exhausted of air. If the point of the instru-ment be not very sharp, it is desirable to make a nick with a scalpel in the skin, pre-viously pulled downwards over the rib below. Then the instrument is thrust boldly into the intercostal space at the acme of an inspiration. Communication is then established with the bottle or syringe, the flow being regulated by the tap or piston, so that the outflow may not be too rapid. Much coughing by the patient indicates that the point is touching the lung. A quantity varying between 5 and 50 ounces may be withdrawn, but the operation must be

stopped if coughing or respiratory distress is caused. Seal the opening with collodion. At the present day some prefer siphonage to the aspirator. If the fluid contain blood, it may denote tubercle, carcinoma or a slight wound of the lung. If it be purulent, the surgical measures for empyema are applicable, and it is wise to be prepared for this eventuality. In cases of serous effusion, it is recommended that the fluid should be withdrawn by siphonage, and replaced by a suitable quantity of sterilised air.

I*a. The physical signs are those of pleurisy with effusion, but it does not clear up in due course, and the patient has* SWEATINGS, SHIVERINGS, *and* IRREGULAR ELEVATIONS *of temperature. The disease is probably* EMPYEMA.

§ 99. **Empyema** is a collection of purulent or sero-purulent fluid within the pleura. It most often follows a serous effusion, but it may be purulent from the beginning.

The *Symptoms* and *Physical Signs* are similar to those of serous effusion (*q.v., supra*), with certain others in addition—viz. : (1) It may be found that the fluid *does not clear up* as a serous effusion should do, and thus the presence of pus may be suspected. (2) Whenever pus forms, either in the pleura or elsewhere, it is marked by the occurrence of sweatings, shiverings, and an intermittent pyrexia. (3) Œdema of the integument, the pointing of an abscess in an intercostal space, over the clavicle, or even in the groin, or copious discharge of pus by the mouth, are in rare instances the first distinct evidence of a localised empyema. (4) The history generally throws considerable light on the case by revealing one of the *causes* of empyema—namely :

(i.) Pneumonia, especially in children, may be followed by empyema [1]; (ii.) septic conditions of the pericardium, mediastinum, or respiratory tract--sepsis in any part of the body may cause a simple effusion to become purulent ; (iii.) tuberculosis in any form in the thorax ; (iv.) the acute specific fevers ; (v.) abscess of the lung—*e.g.*, in bronchiectasis—abscess of the liver or spine bursting towards the pleura, or peri-hepatic abscess resulting from appendicitis, leaking gastric or duodenal ulcer ; (vi.) careless paracentesis, or any wound from without, permitting the introduction of organisms.

(5) In doubtful cases a leucocyte count should always be made, since in the absence of acute lobar pneumonia more than 20,000 leucocytes per cubic millimetre would strongly favour a diagnosis of empyema. (6) The aspiration of a few drops of the fluid with a hypodermic needle will often settle the diagnosis, though there are two fallacies in this method : first, in rare cases the fluid may be too thick to come through the needle ; or, again, the pus may be encysted between the lobes of the lung. In any case, a microscopical examination of the material at the point of the needle may assist the diagnosis.

Prognosis.—Empyema is always serious, and may run a somewhat prolonged course of some months. Cases of pure pneumococcal empyema are much more favourable than those due to streptococci or staphylococci,

[1] In children there is often rapid onset of pus without constitutional signs, as in Pyopericarditis (§ 43).

either alone or with the tubercle bacillus. Its course can be considerably modified by prompt and adequate surgical treatment. Early operation, adequate drainage, and strict aseptic precautions, both at the operation and at the subsequent dressings, are the points in treatment which most favourably influence prognosis. If left to itself, the results vary : sometimes there is compression and destruction of the lung ; sometimes, as above mentioned, the pus opens into the lung, burrows in various directions, or opens through the chest wall ; or it may lead to pyæmia.

Treatment.—When we are sure that the fluid is purulent the empyema should be opened and drained without delay. Every aseptic precaution should be taken. To drain an empyema it is usually necessary to remove 1 to 1½ inches of rib, which is best taken from the seventh or eighth rib in the posterior axillary line. When the patient is anæsthetised, insert a needle in order to locate the pus. This should determine the site of the operation, a point being selected in as dependent a position as possible. The skin is pulled down with the finger, and an incision 3 inches long is made on and parallel to the rib. The periosteum is scraped aside, and a piece of rib is removed with strong bone forceps. The parietal pleura is then incised. The intercostal artery situated just beneath the lower border of the rib should be avoided ; if cut, it must be ligatured. The finger should be introduced into the cavity as soon as it is opened and before the pus has drained away. By this means any adhesions may be gently broken down and the large fibrinous flakes of pneumococcal empyemata be removed. Drainage should be effected by means of a large tube, which can usually be shortened to 1 inch or so after about forty-eight hours. At subsequent dressings the same strict asepsis should be maintained, because secondary infection makes the prognosis much worse. In the case of very large empyemata, causing great embarrassment, it is sometimes advisable to remove some of the pus by aspiration as a preliminary measure, but this should be followed by operation after about twelve hours.

II. *The patient has been* TAKEN ILL SUDDENLY ; *the temperature is high, the dyspnœa considerable, and the expectoration soon becomes rusty ; there are* SIGNS OF CONSOLIDATION *at the base of one lung. The disease is* ACUTE LOBAR PNEUMONIA.

§ 100. Pneumonia—*i.e.*, inflammation of the pulmonary tissue proper, or parenchymatous inflammation—occurs in two forms. The *first* and more acute is, from its area of distribution, termed " Lobar Pneumonia," or, from the nature of the inflammation, " Croupous Pneumonia." The *second* is termed " Lobular Pneumonia," because it affects the lobules of the lungs (also called Broncho-pneumonia, Catarrhal Pneumonia ; see below).

Acute Lobar Pneumonia commences suddenly, with well-marked constitutional symptoms, such as headache, backache, rigor, and, in children, vomiting. The temperature during the rigor rises to 103° or 104° F.,

and it remains at this point for about a week (Fig. 43). The aspect of a pneumonia patient is very characteristic (§ 7)—the face is flushed, and herpes often appears on one side of the mouth. There is pain in the affected side, short cough, shallow, rapid breathing, and on the third or fourth day tenacious rusty-coloured sputum. The pulse-respiration ratio is 2 to 1, instead of the normal 4 to 1. The urine is scanty, high-coloured, with diminution of the chlorides. The patient shows more and more distress, and in a short time there may be delirium, with signs pointing tô failure of the heart. About the *seventh* or *eighth* day the fever, in favourable cases, terminates by crisis, falling to normal in the course of a few hours. This is accompanied by marked general improvement ; the pulse-respiration ratio returns to normal, and a critical sweating or diarrhœa may occur. Crisis often occurs on the odd days—*i.e.*, fifth, seventh, ninth, or eleventh of the disease. Pseudo-crises occasionally occur, but these are distinguished from true crises by the fact that the pulse and respiration do not return to normal. In rare cases the temperature falls by lysis. The whole illness lasts about two or three weeks. If it lasts longer, *tuberculosis should be suspected* (§ 101).

·The *Physical Signs* are limited to one lobe or one lung, usually the right lower lobe. It is only in

FIG. 43.—ACUTE LOBAR PNEUMONIA, showing typical crisis on the seventh day. George H., aged thirty-five, was taken ill very suddenly when in the infirmary with shivering and acute pain in the side.

rare cases that both lungs are affected. Percussion may, for the first day or two, reveal no dulness, but, as a rule, there is elicited early in the disease slight impairment of the percussion note, which soon becomes dull. On auscultation, the breath sounds are weak, and fine rustling crepitations are heard, which have been compared to the rustling of hair or tissue-paper, against the ears. As the inflammatory exudation increases, the lung tissue becomes solid, and over the dull area we get all the *signs of consolidation* (p. 150). When the fever abates, coarse moist râles (redux crepitations) are heard, and the percussion resonance and normal breath sounds gradually return.

Etiology.—Pneumonia occurs at all ages and in both sexes, but is commonest in adult males. It is a microbic disease, the specific cause being

a diplococcus, the pneumococcus of Fraenkel. Debilitating influences, such as exposure, are said to predispose to the disease ; but it is surprising how often strong, apparently healthy men are attacked, and these not infrequently succumb. A blow on the chest may determine an attack. Like other local inflammatory diseases, it may arise as a complication of a constitutional malady ; the acute specific fevers in particular rendering a person vulnerable to the pneumococcus. It frequently heralds the end in chronic maladies such as Bright's disease, cancer and cardiac disease. When pneumonia runs an atypical course we should always bear in mind the possibility of the lung affection being only a complication of a constitutional disease such as typhoid fever.

Diagnosis.—Pneumonia is diagnosed from acute *pleurisy with effusion* by means of the data given in the table of diagnosis between consolidation of the lungs and fluid in the pleura (§ 89). *Broncho-pneumonia* runs a different course, and the signs are scattered over both lungs (see table below). The sudden onset of acute pneumonia resembles that of *scarlet fever, erysipelas,* and *small-pox,* but the absence of rusty sputum and altered pulse-respiration ratio distinguishes them. There is a pneumonic form of *acute pulmonary tuberculosis* which has to be borne in mind (§ 101) ; also various *aberrant forms of pneumonia* (§ 102). Pneumonia may at its onset simulate *abdominal inflammation,* pain being referred to the abdomen, and lung signs being absent (§§ 188 and 193).

TABLE VII.—DIFFERENTIATION BETWEEN

	LOBAR OR CROUPOUS PNEUMONIA.	LOBULAR OR BRONCHO-PNEUMONIA.
Onset	Sudden, with rigors	Gradual, and preceded by bronchitis.
Course of Temperature	Continuous	Remittent.
Defervescence	By crisis seventh day	By lysis in three to four weeks.
Percussion	Dulness in one lung, usually the base.	Scattered patches of dulness in both lungs.
Auscultation	(i.) Fine crepitations (ii.) Consolidation signs in a day or two.	(i.) Fine crepitations and consolidation signs over dull areas, though obscured by rhonchi and bronchitic râles.
Sputum	Rusty	Frothy and muco-purulent.
Respiration	Pulse-respiration ratio 2 : 1.	No marked difference of pulse-respiration ratio.

Prognosis.—The case mortality varies from 20 to 40 per cent. in hospital cases ; the usual cause of death being heart failure. Much depends on the position and extent of the lesion, which is graver when both lungs are involved or when the disease attacks the apex. The reason for this is that apical pneumonia usually occurs in a lung already damaged by tubercle. It must, however, be remembered that small areas of consolidation may be associated with very great toxæmia. A lethal termination

may be anticipated with marked cyanosis, a typhoid condition, scattered râles over both bases (indicating œdema), with lowered temperature. Absence of the usual increase in the leucocytes is of the gravest import. Pneumonia is graver in elderly persons, in alcoholics, and in debilitated persons ; children, from 3–10 years, nearly always recover, but robust men in the prime of life often succumb, although the prognosis is generally stated to be good in healthy adults.   As regards complications, meningitis is generally fatal, and endocarditis extremely grave.   Jaundice, meteorism, and acute gastric dilatation may occur.   But of all conditions influencing the prognosis of lobar pneumonia chronic alcoholism is, in my belief, the worst. Delayed resolution, lasting one to three months, is uncommon ; abscess, empyema, gangrene and chronic pneumonia may supervene in weakly subjects.

Treatment.—There is at present no specific remedy for pneumonia, so that treatment is mainly expectant.   Our chief endeavour should be to maintain the patient's strength, and to achieve this, rest in bed, good nursing, and visits by the doctor at least twice a day are essential.   Fresh air is essential.   Patients treated near an open window have less dyspnœa and cyanosis, and do better than those treated in a vitiated atmosphere. They should be kept thoroughly warm by blankets and hot bottles.   The bowel should be cleared with calomel.   The diet must be fluid, 2 to 3 pints of boiled milk, and whey or barley water, being sufficient for the first few days.   Raw eggs, broths and jellies can be added later.   If the food decomposes, acute dilatation of the stomach and intestinal paresis may set in, with vomiting and abdominal distension ; pituitary extract and lavage will avert a fatal issue.   For meteorism give turpentine ℥ ss. to a large enema.   Saline infusions (500 c.c.) may be given to relieve the toxæmia when the constitutional symptoms are severe.   *Sleep* is of such paramount importance that no patient should be allowed to spend a restless night.   The cause for the restlessness should be sought for and treated. Frequently *pain* is the disturbing factor.   This may be relieved by the local application of ice, fomentation, poultices, a blister, or a leech. Another cause of sleeplessness is *engorgement of the right heart.*   In every case of pneumonia careful watch should be kept over the right heart. If, in the early stage of the disease, the patient is blue and restless, the cardiac dulness increased considerably to the right, the liver enlarged, and the veins of the neck full, we should immediately relieve the right heart, either by venesection (5 to 10 ounces) or by applying six leeches to the skin over the liver.   This extreme condition may be averted by the timely use of two or three leeches.   *Pyrexia* over 103° may be the reason for sleeplessness, and may be reduced by tepid sponging, a measure which next to the relief of pain and engorgement of the right heart is the most satisfactory means of procuring sleep.   For the sleeplessness, hypnotics, such as paraldehyde, chloral, or morphia may be given, but never sulphonal.   Opium (10 gr. (0·6) Dover's powder) may be used in the early stages of the disease, and is often of the greatest value, but if the right

heart shows signs of engorgement, it is better to give morphia with atropine and hyoscine.

General and Cardiac Stimulants.—Strychnine should be injected hypodermically, beginning with ℥ 3 (0·18) of liquor strychninæ every eight hours on the fourth day, gradually increasing the dose according to circumstances, until the crisis is over. Digitalis, to be of use, must be given early and continued. When the blood-pressure is low, Sir Jas. Barr recommends injections of pituitary extract, 1 c.c. every eight hours. Concerning alcohol, there is much difference of opinion. It is particularly' indicated in alcoholic patients, for whom it should be used freely (4 to 12 ounces whisky in twenty-four hours), and especially in conditions of collapse near the crisis, when it may tide the patient over so that he is out of danger before the subsequent depressing effect of the drug becomes manifest. The value of oxygen inhalations is well proved, especially in cases with cyanosis. The most convenient method of administration is by a nasal catheter, two or three litres per minute.

Concentrated oxygen is an irritant, and it should therefore be diluted with air and it should always be warmed by the passage through warm water before reaching the patient. Vaccine, in small doses, may do good if given at the beginning of the disease, but is a remedy to be used with the utmost caution; the initial dose should not exceed 5 million pneumococci. The correct type of pneumococcus must be employed. Antipneumococcic serum is sometimes valuable when given in the early stage. Preventive treatment comprises care of the mouth, teeth, and nasopharynx, pneumococcal and influenzal vaccines.

§ 101. **A Pneumonic Form of Acute Pulmonary Tuberculosis,** or pneumonic phthisis, is often met with. The symptoms resemble those of pneumonia, and may start suddenly with a rapid rise of temperature and pain in the side. The temperature may continue high for a week or so. The physical signs also resemble those of pneumonia. It differs from this disease, however, in the presence of tubercle bacilli in the sputum, and the temperature, instead of falling abruptly by crisis about the seventh day, gradually becomes intermittent, and the *course of the disease* becomes indefinitely prolonged for weeks. This is followed by physical signs of breaking down, purulent expectoration, night sweats, and in some cases death in five to twelve weeks from exhaustion, hæmoptysis, or complications, such as pneumothorax (§ 104).

§ 102. **Aberrant Acute Pneumonias** (Deuteropathic Pneumonia).—We have seen that in pleurisy, acute lobar pneumonia, and in other inflammatory diseases of the lungs, the course of the malady is fairly definite, and the physical signs in the lungs are characteristic. But it is important to remember that these same conditions may · occur secondary to, or as part of, some general disorder. Under these circumstances some of the symptoms or physical signs may be wanting or irregular, and it may not be possible to arrive at a diagnosis, except by passing in review the whole history of the case, and by making a thorough and systematic examination of all the other organs. Instances of this eccentric group of pneumonias are met with in acute glanders, plague, anthrax, syphilis of the lung, actinomycosis, and psittacosis.

The practical outcome of those conditions is that when a case of pneumonia, or other apparently local inflammatory condition, is *atypical* in its physical signs or its clinical history, we probably have to do with a manifestation of one of the conditions just mentioned, or some general disease, such as enteric fever, influenza, scarlatina, pyæmia, or other general infective disorder.

III. *The illness has come on somewhat* GRADUALLY ; *there is cough, with*

frothy expectoration ; the physical signs of CONSOLIDATION *are* SCATTERED *and accompanied by signs of bronchitis. The disease is probably* BRONCHO-PNEUMONIA. .

§ 103. **Acute Lobular Pneumonia,** or Broncho-pneumonia (catarrhal pneumonia), is also an acute parenchymatous inflammation of the lungs, but it runs a very different course to that of acute lobar pneumonia. The inflammatory process occurs in small patches, scattered unequally throughout both lungs, and it is accompanied by bronchitis : hence its name.

• The *Constitutional Symptoms* come on more gradually in this disease. The temperature is remittent, about 100° F. in the mornings and 101° to 103° F. in the evenings, accompanied by cough, dyspnœa, and frothy sputum. The pulse is rapid, but the pulse-respiration ratio is not altered to anything like the extent of that in lobar pneumonia, and the face is generally pale instead of flushed. The fever is maintained by the fresh implication of neighbouring lobules for about three to six weeks or longer.

Physical Signs.—When the patches of consolidation are small, there may be no dulness on percussion, but only tubular breathing ; but when they are of moderate size, signs of consolidation (§ 89) can be made out. The chief auscultatory signs in children consist of *intensely loud, "* consonating," rhonchi and ronchi.

Etiology.—Broncho-pneumonia occurs at all ages, but is *especially frequent in young children.* The cases fall into two groups, primary and secondary. Primary broncho-pneumonia, due to the pneumococcus, arises in much the same way as lobar pneumonia. Secondary forms arise : (i.) Complicating acute infections, such as measles, whooping-cough, diphtheria, small-pox, influenza, typhoid and scarlet fevers ; (ii.) complicating chronic debilitating conditions, such as chronic Bright's disease, chronic cardiac disease, or bed-lying, as from fracture of the femur in old people ; (iii.) *aspiration* or *deglutition (septic) pneumonia,* such as occurs after operations on the tongue, mouth, or nose, in quinsy, cancer of the œsophagus communicating with the air-passages, bronchiectasis, and following hæmoptysis or the passage of food down an insensitive trachea, as in post-diphtheritic paralysis. If cleanliness of the mouth and naso-pharynx is ensured before operation in that region this type of pneumonia cannot develop. A common but more chronic variety is of tuberculous origin.

Diagnosis.—*Chronic phthisis* is limited to the apex at first, and runs a characteristically chronic course. The pulmonary signs of *measles, whooping-cough,* and *bronchitis* resemble broncho-pneumonia in its early stages, and it may be easy to diagnose these several diseases until the rash of the one or the whoop of the other appears. The constitutional symptoms in acute bronchitis are much less severe. The diagnosis from *acute miliary tuberculosis* may be very difficult, as sputum is usually not obtainable. The diagnosis from *lobar pneumonia* is given in tabular form above (p. 166).

Prognosis.—The case mortality in children under five varies from 30 to 50 per cent. (Osler) ; the younger the child the more fatal is the disease. The strength of the patient and the duration of the disease are leading factors in the prognosis. If he is debilitated, especially if the environment is unfavourable, he soon becomes a prey to the tubercle bacillus,

and the case rapidly runs on to phthisis (*q.v.*). Similarly, the longer the case lasts the more likely it is to have a fatal termination, and in adults this is very often the case. Broncho-pneumonia is nearly always secondary, and the third leading factor in the prognosis is the nature of the antecedent disease. When a child weakened by a *prolonged* fever is attacked, the prognosis is very grave, but after whooping-cough and measles it is more favourable. Nevertheless, children often recover in apparently hopeless cases. The aspiration and deglutition pneumonias are usually fatal.   •

Treatment resembles that of lobar pneumonia, but stimulants are indicated from the outset of the disease ; two drops of brandy for every month of an infant's age may be given every second hour. Children should be placed in a steam-tent, and small frequent doses of tr. belladonnæ administered. For adults the pain and incessant cough may require opium, best given as Dover's powder, and poultices applied to the back give considerable temporary relief. The chest afterwards may be covered with a cotton-wool jacket. If the symptoms become more distressing and the cough and dyspnœa increase, stimulating expectorants should be ordered ; and if the cough continue difficult an emetic may be given. For the reduction of the hyperpyrexia, cold sponging may be adopted, especially if cerebral symptoms are present. When a mixed infection is present, appropriate vaccine or serum should be used. Success has been obtained with antistreptococcus serum in cases with streptococci in the sputum.   •

We now turn to the **acute disease with hyper-resonance on percussion** —viz., Pneumothorax. We must bear in mind that an acute disease may supervene upon a chronic condition accompanied by hyper-resonance —*e.g.*, when acute bronchitis supervenes on emphysema (see Table IX., § 120).

The patient is in MARKED DISTRESS, *which has come on* SUDDENLY ; *there is often hyper-resonance and usually absence of breath sounds. The disease is* PNEUMOTHORAX.

§ 104. **Pneumothorax** is a term used to denote the presence of air in the pleural cavity, the air having gained admission by perforation of the pleura, either from within or from without. The air is after a time accompanied by pus, and the condition is then known as pyopneumothorax ; if accompanied by serous effusion, as hydropneumothorax (Fig. 44).

The *Symptoms* of the onset of the condition differ according to the condition of the lung—*i.e.*, whether it is fairly healthy or is widely diseased. (*a*) When pneumothorax occurs in the less affected of the two lungs—the other side being extensively diseased—the symptoms are very urgent, and consist of severe pain in the side, attended by great dyspnœa, shallow, quick breathing, cyanosis, and some degree of collapse, with sweating, lividity, and a weak pulse. The patient usually lies on the healthy side. (*b*) In other cases, where pneumothorax comes on in a lung which is already much diseased, the onset may be hardly noticed.   •

The *Physical Signs* consist of : (i.) A bulging on the affected side ; (ii.) diminished vocal fremitus ; (iii.) hyper-resonance on percussion (unless there is very great distension, when the note may be dull) ; (iv.) on auscultation the respiratory murmur

may either be inaudible or amphoric ; the vocal resonance is usually diminished, but pectoriloquy and bronchophony are sometimes present. The *bell sound* may be elicited on tapping the chest with two coins in one position, and listening with a stethoscope in another. When fluid is also present, and this is usual, metallic tinkling is heard. The *Succussion Splash*, when it can be elicited without damage to the patient, is the most characteristic sign of hydropneumothorax—a fact which was well known to Hippocrates.[1] It may be heard by placing one's ear against the chest whilst shaking the patient's body to and fro.

Etiology.—(i.) Undoubtedly the commonest cause (75 per cent.) is advanced phthisis, when a cavity bursts into the pleura. (ii.) The converse process may take place in empyema, when the pus bursts into the lung. (iii.) A fractured rib may lead to perforation of the lung. (iv.) Less common causes are gangrene of the lung, abscess connected with the spine or liver, or an ulcer of the stomach or oesophagus, bursting into the pleural cavity. (v.) It rarely happens in healthy people, although cases have been recorded.[2]

Prognosis. — The occurrence of pneumothorax is always very grave. It is difficult to estimate its case mortality, because death may be sometimes produced by the condition of the lung apart from the accident, but about half die within the first week, and some in a few hours, from shock or suffocation, when the lung on which the patient has been mainly dependent gives way. Only about 10 per cent. of all cases taken together ultimately recover. The *immediate* risk depends upon the urgency of the dyspnœa and cyanosis, the state of the other lung, the patient's general health, and the cause of the accident. As regards the *cause*, the pneumothorax that results from late phthisis or gangrene

Tympanitic per-cussion ; absence of breath and voice s o u n d s, and of v o c a l resonance.

Percussion d u l-ness ; metallic tinkling ; splash on shaking (suc-cussion).

FIG. 44.—DIAGRAM OF HYDROPNEUMOTHORAX.

of the lung is very fatal ; but that which occasionally complicates whooping-cough, pneumonia, early phthisis, and injury, often results in recovery. Certain it is that the longer the patient lives after the onset of the pneumothorax, the better is the prognosis for ultimate recovery (p. 184). Death usually occurs from shock and suffocation, as mentioned above, or from asthenia, due to the prolonged discharge and lung disease.

Treatment.—A hypodermic of morphia is desirable for the pain, and stimulants for the collapse. The question of paracentesis for the removal of air is important. It has been done with advantage when great distension is present, as indicated by marked displacement of organs, extreme pain and discomfort, but the relief is usually

[1] " De Morbis," lib. ii., cap. xvi.
[2] Transactions of the Medical Society, 1897, vol. xx., p. 120.

only temporary. When pus is present, the treatment should be that of empyema. If clear fluid is present, it may be aspirated. In phthisical cases it is often inadvisable to aspirate or operate ; great improvement usually follows, because the lung is rendered inactive, and is in a state of passive congestion.

There is one disease of the lungs which belongs neither to the acute nor to the chronic category, but is PAROXYSMAL, *occurring in attacks of sudden onset, usually* WITHOUT ELEVATION OF TEMPERATURE—ASTHMA.

§ 105. Asthma is characterised by paroxysmal attacks of very severe dyspnœa, the inspiratory effort being short, the expiratory prolonged. It is accompanied by much lividity and distress. Chronic bronchitis is liable to complicate asthma, but it is important here to draw attention to the frequent error which is made by regarding exacerbations of chronic bronchitis as paroxysms of asthma.

Symptoms and Clinical History.—The leading characteristic of this disease is its paroxysmal nature. A person who is subject to asthma may be perfectly well at one minute, and half an hour later may be seized with the most violent dyspnœa. An attack often commences in the early morning, the patient awakening with a feeling of tightness of the chest ; he gasps for breath, and clings to surrounding objects in order to bring into play the extraordinary muscles of respiration. Each attack lasts from a few minutes to a few days, and then, without apparent reason, the patient rapidly recovers his normal and healthy condition.

There are many curious and unexplained features in connection with this malady, one of the most interesting being the tendency to skin eruptions (especially urticaria and the various forms of erythema), and another the fact that these eruptions may alternate with the attacks of dyspnœa. Various other neuroses, and even attacks of insanity or epilepsy, may alternate in the same way. The paroxysms of asthma are occasionally preceded or ushered in by violent attacks of sneezing, by itching, or by the passing of large quantities of limpid urine. Sometimes an attack is terminated in this way.[1]

Physical Signs.—On inspection the chest is seen to be maintained in a position of inspiration, undergoing but little expansion with the short inspirations. The percussion note may be unaltered, but, after many attacks, emphysema supervenes, with consequent hyper-resonance. On auscultation the short inspiratory effort is feeble and scarcely audible ; expiration prolonged. Loud rhonchi and often coarse râles replace the normal vesicular murmur, owing to the accompanying bronchitis.

Etiology.—Some regard the asthmatic attack as a series of spasmodic attempts on the part of the respiratory muscles to overcome some obstruction to the entry of air. But the central fact, which alone explains all the symptoms, is a narrowing of the bronchial tubes, probably due to spasm of the involuntary bronchial muscles, with hyperæmia of the submucosa and swelling of the mucous membrane. Some hold that the disease is therefore an angioneurosis. Recent investigation points to asthma often being a manifestation of anaphylaxis. A foreign protein enters, perhaps in the food, perhaps from some infective focus in the nasopharyngeal passages.

Among the *predisposing* causes we find : (i.) A neurotic family history. Careful inquiry may reveal asthma or other neuroses, especially those so-called functional diseases of the nervous system connected with the involuntary muscular system, such as attacks of flushing and shivering, faints, and the like. (ii.) Asthma may occur at any *age*, but nearly always makes its first appearance soon after the age of puberty. (iii.) Any previous lung disease, especially chronic bronchitis, may predispose to asthma. Malaria, gout, and other constitutional conditions, are often associated with it. (iv.) Conditions of the nasal passages, such as ulceration, hypertrophic rhinitis or polypi.

Among the *exciting* causes of an attack may be mentioned : (i.) Certain atmospheric conditions which are ill-understood, and often appear to be most contradictory. Thus

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[1] These facts point possibly to an infection of the general vaso-motor system similar to that in the pulmonary system which produces the spasmodic dyspnœa.

I know one patient who is free from asthma in London, but develops an attack immediately she seeks a high altitude. Another always develops an attack when she enters London. Some find the sea relieves them, others that a seaside place determines their attacks. (ii.) Reflex causes, such as derangement of the alimentary canal, and dietetic indiscretions; (iii.) dust and irritating particles; (iv.) conditions as in hay fever, *e.g.*, proximity to horses, or certain plants.

Diagnosis.—The diagnosis usually presents no difficulty. The paroxysmal occurrence of the disease is quite characteristic. Paroxysms of dyspnœa coming on at night occur in the course of Bright's disease and cardiac disease, and have been loosely called asthma. The actual toxins producing asthma may often be determined by performing a series of vaccinations (as in Von Pirquet's test) with solutions of the proteins of different substances, *e.g.*, rye, wheat, eggs, feathers, grass, etc., and thus certain definite indications for treatment may be obtained.

Prognosis.—The disease of itself does not shorten life, but tends to produce emphysema, bronchitis, and their attendant evils. Children may grow out of the disease; adults never lose it completely. The severity and frequency of the attacks are our only guides to prognosis.

Treatment.—(*a*) *During the Attack.*—Various remedies have been tried. Mentioned in the order in which I have found them most useful there are : tr. lobelia, belladonna, hyoscyamus, opium in small doses, and pyridin, a remedy introduced and strongly advocated by Germain See. An injection of adrenalin (1 in 1,000) may abort an attack. The earlier the drug is given, the smaller need be the dose; even one drop may abort an attack. Spraying the nose with the solution is often efficacious. Atropin and cocaine sprays also relieve. The diet during the attack should be the lightest possible; milk alone is best. Various inhalations are useful, for the prevention or relief of an attack—*e.g.*, the vapour from a teaspoonful of turpentine and chloroform, or the fumes of paper prepared with a strong solution of nitrate of potash, or amyl nitrite. If a mixture containing equal parts of the leaves of stramonium, lobelia, black tea, and potassium nitrate be burnt in a tin plate, and the fumes be inhaled, much relief is usually afforded. Various other preparations, in the form of cigarettes of stramonium, potassium nitrate, and belladonna, are used.

(*b*) *Between the Attacks.*—The effect of locality on the disease can only be ascertained by experience. As a rule, though with many exceptions, town air and fogs are detrimental. To prevent an attack, special attention should be directed to the diet. Avoid solid meals after two o'clock in the day. Iodide of potassium, administered for a long period of time, certainly tends to ward off attacks in some patients, and potassium bromide at bedtime may act similarly. Arsenic is also useful. The nose should be examined for polypi, etc., and these must be removed. All sources of reflex irritation must be sought for and treated. Injections of normal horse serum (given every fourth day in doses of 4 minims increasing to 2 c.c.) will cure the asthma due to proximity of horses. Working on the anaphylactic theory, Auld has found peptone acts as an immunising agent in asthma. He injects ⅓ gm. peptone (Armour) dissolved in 5 c.c. distilled water at body temperature, two doses a week, and gradually increases to 1 gm. In some cases benefit may result from the use of an autogenous vaccine containing small doses of all the intestinal flora.

CHRONIC DISEASES OF THE LUNGS AND PLEURÆ

106. Classification.—Chronic disorders of the lungs and pleuræ may follow an acute attack of the conditions described in the previous sections, as when chronic bronchitis and emphysema succeed attacks of acute bronchitis. But many of the chronic diseases of the lungs, such as pulmonary tuberculosis, start insidiously, and attention may not be directed to the lungs for a considerable time.

The chronic diseases, like the acute, may be classified for clinical purposes, according to the results of percussion. It is convenient in actual

practice, although unscientific, from the point of view of classification, to make a subsidiary group in which the sputum is highly offensive or has some other characteristic feature.

§ 107. Method of Procedure.—The routine examination is conducted as in acute disorders (§ 93)—viz., after ascertaining the leading symptom, and the history of the illness, we proceed to Inspection, Palpation, Percussion, and Auscultation. In percussion, remember to keep the hand flat and firmly pressed against the chest, while it is struck by the tips of the fingers of the other hand, used as a hammer, and with a staccato stroke. Remember also that the note is normally dull over the mammæ in most women, over the scapulæ in muscular men, and that it is slightly lower-pitched at the right than the left apex. The chest must, of course, be stripped.

GROUP A.—The patient's symptoms point to **chronic disease of the lungs,** and on examining the chest there is **no alteration in the percussion note.**

I. *The patient has a chronic cough ; there is no elevation of temperature, and on auscultation* RHONCHI *and* RÂLES *are heard over the chest. The disease is* CHRONIC BRONCHITIS.

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[1] Pneumothorax sometimes comes on acutely, but it is more often part of a chronic disease.

§ 108. **Chronic Bronchitis** is a chronic inflammation of the bronchial tubes. It may be chronic from the beginning, or it may supervene on repeated attacks of the acute disorder.

Symptoms.—A patient with chronic bronchitis and—its usual sequel —dilated right heart, presents a typical appearance. Stout in build, with short, thick neck, of florid complexion, short of breath, wheezy respiration, and pulsating jugular veins, he presents an aspect which can be recognised at once. The clinical history extends over many years, with alternate diminution and aggravation of the symptoms. The cough is usually present during the winter, and improves as the weather gets warmer. The constant coughing and straining to bring up the secretion results sooner or later in generalised emphysema. In later stages the cough continues all the year round, and finally an attack of capillary bronchitis, œdema of the lung, or some intercurrent malady, throws a little extra strain upon the overburdened right heart, and death ensues. There are, as a rule, no febrile or constitutional symptoms.

The *Physical Signs* vary with the amount of secretion present, the extent of the complicating emphysema (§ 120), and bronchiectasis (§ 121). In cases of long duration the chest is barrel-shaped (emphysematous, § 86). Rhonchial fremitus may be felt on palpation. On percussion there is never any dulness, and the note is hyper-resonant in proportion to the emphysema present. On auscultation sibilant and sonorous rhonchi and bubbling râles can be heard ; and crepitations at the base, due to œdema, may be present.

There are four recognised varieties of this disease : (i.) *Bronchitis* with *winter cough*, attended by slight or abundant expectoration, mucous or muco-purulent, sometimes fibrinous, sometimes containing streaks of blood. (ii.) *Dry Bronchitis* is attended by a frequent cough and soreness of the chest, but little or no secretion ; it is of a very obstinate character, and occurs mostly in elderly people of a gouty diathesis. (iii.) *Bronchorrhœa* is recognised by the expectoration, which is of a thin, clear, or thick and ropy nature, very abundant and devoid of air. (iv.) *Fœtid Bronchitis* may occur in the later stages, and marks the onset of bronchiectasis. The sputum is very fœtid from time to time (see Bronchiectasis, § 121). (v.) *Plastic Bronchitis*, described § 109.

The *Diagnosis* of chronic bronchitis is not usually difficult. It may be readily diagnosed from *chronic phthisis* by the appearance of the patient, by the absence of hectic fever and emaciation, and by the absence of the tubercle bacillus from the sputum.

Etiology.—Chronic bronchitis may occur at any age, but is more common in elderly people. Sometimes, as before stated, it follows repeated attacks of acute bronchitis, but it may be chronic from the beginning. It often affects plethoric subjects, especially those of a gouty habit, and it is one of the recognised complications of Bright's disease. It is a frequent sequel to cardiac valvular disease, more especially disease of the mitral orifice. It may complicate other diseases of the lungs, especially

phthisis, and may be a sequel of the acute specific fevers, especially measles and enteric fever.

Prognosis.—Patients with chronic bronchitis seldom entirely recover, though they may live for a great many years ; and if the heart is fairly healthy and care be taken to avoid exposure, life is not very materially shortened. The coexistence of gout, Bright's disease and cardio-vascular degeneration make the prognosis somewhat less favourable. The condition of the lungs is not so much a guide to prognosis as the condition of the heart. This, indeed, is the point around which the prognosis centres, and the untoward symptoms which render the prognosis grave are thus referable to the heart—viz., considerable dilatation of the right heart with evidences of cardiac failure, such as dropsy, rapid, irregular pulse, great breathlessness, and cyanosis (see § 55).

Treatment.—The extreme frequency of the disorder renders the treatment a matter of considerable importance. In severe cases the patient must be confined to one room at a uniform temperature of 62° F. day and night. When the mucous membrane is dry and irritable, a steam kettle gives great relief ; it must be kept constantly going, not used intermittently. In slight cases, however, the patient can go about, but chill and exposure should be avoided. The important 'question of when a patient may go out must depend largely on the weather—cold and moisture, especially when in combination, are especially injurious.
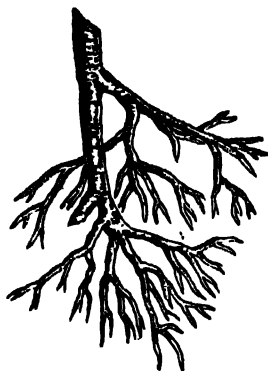
FIG. 45.— BRONCHIAL CAST.

The indications as to treatment are : (i.) To stimulate the relaxed mucous membrane with such remedies as am. carb., senega, squills, etc. (ii.) When the cough is dry, soothing remedies, such as bromides, codëia, and tr. camph. co. should be given, or remedies directed to promote the secretion, such as ipecac., ammon. chlor., potass. iod., and alkalies (the last two especially in rheumatic or gouty cases), may be employed. (iii.) When the sputum is very abundant, we should endeavour to diminish secretion by such remedies as the balsams (tolu and peru), tar preparations (creosote, guaiacol, petroleum), turpentine, camphor, senega, etc., given either internally or in the form of inhalations. For the latter, tar, creosote, and terebene may be used. Counter-irritants to the chest— *e.g.,* turpentine, camphor, or eucalyptus, are very popular with some. (iv.) When there is much spasm of the tubes, lobelia, iodide, and other remedies for asthma are to be tried. (v.) Cardiac tonics and stimulants are called for sooner or later where dyspnœa and other cardiac symptoms are present. (vi.) In cases with a gouty taint Ems water each morning, and small doses of iodide may be added to the other treatment, and the emunctories may be aided by sipping hot water morning and evening,

and at intervals during the day. (vii.) In the fœtid type antiseptic sprays and intratracheal injections with a special syringe after anæsthetising the larynx.

§ 109. **Plastic Bronchitis** is inflammation of the bronchi, with the formation of fibro-plastic casts, which are expectorated.

Symptoms.—The symptoms consist of (i.) violent attacks of coughing, with expiratory dyspnœa, followed by (ii.) the expectoration of a fibrinous cast of a bronchus (*vide* Fig. 46). (iii.) The patient generally suffers from chronic bronchitis, and a little hæmoptysis may follow the expulsion of a cast. (iv.) Sometimes there are no constitutional symptoms, but slight pyrexia, and in some cases even rigors may be present. Such symptoms supervening in a case of chronic bronchitis lead us to suspect the condition.

Physical Signs may be absent. If present, they are those of an obstructed bronchus —an absent or diminished respiratory murmur, accompanied possibly by impaired percussion note. Whistling rhonchi or " flapping " sounds may be heard.

Causes.—The disease is twice as common in men as in women. It may occur at any age in subjects of chronic bronchitis.

Prognosis.—The condition is more serious than bronchitis. Two varieties have been described : (1) An acute form, lasting for some weeks ; and (2) a chronic form, occurring at intervals, for years, in the course of chronic bronchitis. Each attack may last for some weeks, and the casts be coughed up daily. The condition occasionally leads up to a fatal issue from dyspnœa, as when a large cast cannot be brought up.

The *Treatment* differs but little from that of bronchitis. The removal of the membrane may be promoted by the inhalation of lime-water, atomised by means of a spray, which is used with a view to dissolve the mucin in the cast. Various oils (*e.g.*, creosote oil, 1 in 40) have been injected as solvents, but the results have not been very promising.

GROUP B.—We now turn to those chronic diseases of the lungs which are accompanied by **dulness on percussion.** (*a*) The more *common* diseases in which the dulness occurs in regular and fairly DEFINED AREAS at base or apex, are : 1. CHRONIC PULMONARY TUBERCULOSIS ; II. HYDROTHORAX ; and III. PULMONARY CONGESTION OR ŒDEMA.

·I. *The patient complains of gradual emaciation and perhaps cough ; on examination of the chest* SIGNS OF CONSOLIDATION *may be found, most marked at the* APEX *of the lung ; there is* INTERMITTENT PYREXIA, *and the sputum may contain the tubercle bacillus. The disease is* CHRONIC PULMONARY TUBERCULOSIS (*Phthisis*).

§ 110. **Chronic Pulmonary Tuberculosis** (Phthisis) may be defined as a wasting disorder due to tuberculosis of the lungs. The word phthisis is objectionable because it only indicates one of the symptoms—viz., the wasting ($\phi\theta\ell\nu\omega$, to waste). In view of the fact that this disease is the chief cause of death in Great Britain (483,321 in 1910), the importance of the subject cannot be overestimated. The number of deaths in 1910 in London alone was 5,555 or 1·14 per 1,000 living. The disease was formerly regarded as due to round, nodular growths, " tubercles," scattered throughout the lungs, which are made up of a large number of small round cells, epithelioid cells, and giant cells. Owing to the discoveries of Koch we now know that these little nodules are only the

inflammatory manifestations consequent on the irritation of a bacillus (the tubercle bacillus), and that the disease is primarily due to the ravages ʼin the economy of this bacillus, and its toxic products, and secondarily, to the supervention of other infective processes, and especially those due to pyogenic organisms invading the lungs.

It is customary to describe the anatomy in three stages. It is now generally believed that tuberculosis of the lungs begins as a tuberculous endo-bronchitis, due to the settling of the microbe in one of the smaller bronchial ramifications. It has been shown by Birch-Hirschfield, who took metallic castings of the bronchial tubes, that the reason the microbe settles at the apex is because in this situation there is, as it were, a " dead end," in which air is not so readily changed as in other situations. As a consequence, any dusty particles containing the bacillus which are inhaled and reach this situation, settle down, and there set up an irritation, resulting in a small localised ulceration of the mucous membrane. This corresponds with the generally accepted teaching that pulmonary tuberculosis is chiefly caused by the inhalation of tubercle bacilli. The *congestion* which takes place around the primary foci constitutes (*a*) the first stage. (*b*) In the second stage masses of cell infiltration fill up the air cells in the neighbourhood of the primary mischief. This is the stage of *consolidation*. (*c*) The third stage is one of *breaking down*. Owing to the indolent character

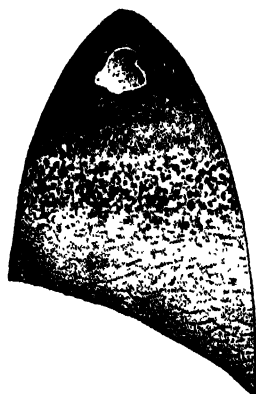III. Cavitation.

II. Consolidation.

I. Congestion.

FIG. 46.—THE THREE STAGES OF PULMONARY TUBERCULOSIS, SOMETIMES PRESENT TOGETHER IN THE SAME LUNG.

and low vitality of the new cell formation, it caseates and softens, becomes the seat of pyogenic organisms, and destruction of the air cells and formation of smaller or larger cavities results. Thus we have three stages : (*a*) Congestion ; (*b*) consolidation ; and (*c*) breaking down, with the formation of cavities (Fig. 46).

Symptoms.—The disease is essentially a chronic one, and its onset is very insidious. It is always more amenable to treatment in the early, stage, and since the introduction of modern methods of treatment an early recognition of the disease has come to be of paramount importance.

(*a*) *Prodromal Stage.*—Phthisis has six modes of onset, which, in order of frequency, are as follows : (i.) Progressive weakness, attended perhaps by cough ; (ii.) hæmoptysis [1] ; (iii.) dyspepsia ; (iv.) laryngeal tuberculosis ; (v) dry pleurisy ; (vi.) acute pneumonia (§ 101), bronchitis, or

[1] Early hæmoptysis of a very profuse kind may occur before any physical signs are discoverable.

broncho-pneumonia. Among the earlier *general symptoms* which should
• make us suspect the invasion of tubercle are unexplained debility, attended
by languor and anæmia on the one hand ; or loss of weight, with unex-
plained dyspepsia, or slight elevations of temperature in the evening on
the other. The temperature is an indication of the very greatest import-
ance, for *no* ACTIVE *tuberculous process can take place in any part of the
body without the occurrence of some pyrexia,* however slight. The type of
this pyrexia is equally distinctive, for it is of an *intermittent character,*
being normal in the morning, and raised in the afternoon or at night ;
in rare instances this is reversed. If we have any suspicion of tubercle,
the temperature should be taken every two hours, so that we may not
miss any slight access of temperature during the day. Allbutt has pointed
out that a premenstrual elevation of temperature sometimes occurs. In
the early stage the patient may not be aware of the feverishness, though
generally he feels a chilliness in the evening, and as the disease progresses,
night sweats form one of its most characteristic features.

The later symptoms of the disease are largely due to the action of
organisms other than the tubercle bacillus. The clinical manifestations
of these " mixed infections " are not always obvious, but many hold that
whenever the temperature rises above 100·5° or 101° F. it is due to a
superadded•infection of this kind. It adds considerably to the gravity
of a case, and its prevention, by fresh air, cleanliness, and a hygienic
mode of life is important.

The *Physical Signs* accompanying the prodromal stage are necessarily
somewhat vague and difficult to detect. The patient's chest should be
thoroughly stripped, and he should be taken to a room where perfect
quiet prevails ; and if with the above symptoms we find weak or unduly
harsh breathing and prolonged expiration at one apex—especially if this
is accompanied by an occasional single sibilant râle—we may be fairly
certain that the disease is developing. Persistent inspiratory " sticky "
clicks at one apex are very suggestive, but not pathognomonic of early
phthisis. It is important to auscultate while the patient coughs, for râles
not previously audible may thus become evident. The signs just named
can often be heard best at the apex, behind, by placing the patient's
hand on his opposite shoulder and listening to that part of the lung, just
external to the bronchi, which will thus be *uncovered by the scapula.* Fine
crepitations may be heard in that situation weeks before any signs may
be discovered at the apex in front. In front the earliest signs may be
heard just beneath the clavicle. Sometimes, later on, we are led to detect
phthisis by an undue loudness of the *heart* sounds at the apex of one lung.
Absence of dulness, like the absence of bacilli, is not evidence of the
absence of tubercle. The *sputum* should be repeatedly examined for
tubercle bacilli. If primary disease of the upper air passages be excluded,
the presence of tubercle bacilli in the sputum is diagnostic of pulmonary
tuberculosis. The early morning sputum should be examined, as it is
the most likely to contain the bacilli. However, the absence of bacilli,

even after several examinations, does not always indicate the absence of phthisis. X-ray examination by an expert is always advisable.

Other tests for the presence of tuberculosis are :

(i.) In *Moro's* test an ointment impregnated with tuberculin is rubbed into the skin ; in tuberculous cases a papulo-pustular rash appears and lasts several days.

(ii.) In *Von Pirquet's* cutaneous reaction the arm is lightly scarified, and a drop of Koch's concentrated old tuberculin is rubbed on the scarified area. In a positive reaction a red papule appears, occasionally after a few hours, usually within twenty-four hours, but it may be delayed even longer. This test is valuable in children ; in adults in cities 90 per cent. of apparently healthy persons react.

(iii.) In *Calmette's* ophthalmic reaction a drop of 1 per cent. of Koch's old tuberculin is placed on the conjunctiva. This test is not devoid of risk to the eye, and is rarely used now-a-days.

(iv.) In the *subcutaneous* tuberculin test Koch's old tuberculin is inoculated under the skin in increasing doses—⅒ milligramme, 1 milligramme, 5 milligrammes, and 10 milligrammes. A positive reaction is shown by (1) a local reaction at the site of inoculation, which is of no importance ; (2) subjective malaise and fever, and (3) reaction at the site of the tuberculosis. Thus in pulmonary tuberculosis crepitations and increased expectoration would occur. This test should never be employed when patients have fever or other obvious organic disease.∪

Dr. Inman points out that none of these tests are of assistance in deciding whether tuberculosis is active or dormant, and they are valuable only as one of many factors which have to be considered before advising lengthy courses of treatment.

(v.) A positive *opsonic index* test, however, points to the presence of active tuberculosis ; but a negative test does not disprove the existence of tuberculosis. The normal opsonic index range is between 0·8 and 1·2. An abnormally high, or an abnormally low, or a widely varying index, indicates the presence of active tuberculosis.

(vi.) The Arneth Count :—A marked shift to the left of the Arneth Count is stated to have considerable positive significance in cases of suspected pulmonary tuberculosis.

(*b*) The *stage of consolidation* and (*c*) the *stage of softening* and cavitation may be dealt with together. The symptoms, physical signs, and the corresponding lung changes are given in the form of a table for the purposes of convenience. The physical signs usually begin at the apex, and are generally best heard at the back, sometimes at the apex of the lower lobe. From this position they extend downwards, and thus it is possible in the same patient to recognise in advanced cases the signs of the third stage, or cavitation, at the apex ; below these, signs of consolidation ; and below these, signs of congestion (as in Fig. 46). Such a condition indicates considerable activity. Many accessory signs may be mentioned : enlarged heart area due to retracted lung, hæmic heart murmurs due to anæmia, clubbed fingers in chronic cases of long duration, etc. Extensive tuberculous disease may sometimes exist with but little constitutional disturbance, and, on the other hand, considerable disturbance of health may be present, without any abnormal physical signs—depending, partly, on the distance of the lesion from the surface of the lung.

The presence or absence of a CAVITY is in the majority of cases impossible to diagnose with certainty. The percussion note is usually dull, but varies with circumstances. Thus the note is resonant when (i.) the cavity is large, or lies very superficially ; and (ii.) there is not a great amount of consolidated lung tissue between the cavity and the chest wall. When the cavity is larger and superficial, and the

communicating bronchus remains patent, a characteristic note, almost tympanitic, is obtained on percussion whilst the patient keeps his mouth open. This is known as the "cracked-pot" sound (*bruit de pot fêlé*). Many attribute most importance to the sign known as "post-tussic suction." To elicit this sign, the stethoscope is applied over the suspected cavity, the patient is told to cough, and immediately after the cough a characteristic swishing sound is heard, due to the sucking of air into the cavity. This may be accompanied by copious râles. The R.M. is often very feeble.

The *Diagnosis* of the disease is not difficult except in the early stages, and in the absence of bacilli in the sputum. (i.) Various other causes of hæmoptysis may have to be differentiated (see § 84) ; (ii.) various other causes of anæmia may have to be eliminated (Chapter XVI) ; (iii.) when the condition begins with dyspepsia, it is very liable to be overlooked until the physician is aware of this mode of commencement ; (iv.) other causes of cough (§ 81) ; and (v.) various pharyngeal and laryngeal affections may have to be excluded (§ 138). (vi.) When it supervenes on bronchitis or broncho-pneumonia, our only clue consists in a delayed convalescence, together with the persistence of râles ; and the fine clicking râles of phthisis are quite distinctive to the experienced ear. In the later stages the differentiation from the other cases of percussion dulness is not difficult (table, § 106).

Etiology.—In phthisis, as in other microbic disorders, there are, on the one hand, predisposing causes which relate to the patient (*i.e.*, the soil on which the bacillus grows) and his powers of resistance ; and on the other hand, exciting causes which relate to the microbe itself. If the " soil " is not suitable—*i.e.*, if the person is not predisposed by heredity or other cause, the bacillus will rarely grow. For these reasons its infectivity has been overlooked all these years. (1) Heredity is a potent cause, the individual being born with a predisposition to the disease.‧ This factor, however, does not, as we shall see, occupy the prominent position which it was formerly believed to occupy. In a large proportion of cases no evidence of heredity is obtainable. (2) Both sexes are pretty equally affected, and the age at which the disease usually supervenes is between sixteen and thirty. The patient may be attacked at any time of life, although it is very rare under two years. (3) Any condition of malnutrition may produce a predisposition to the bacillus invasion, whether it arise from deficient food, from hyper-lactation, from exhausting diseases such as diabetes, or the acute specific fevers, after which an attack of phthisis is by no means infrequent. It is a curious circumstance that pregnant women are not prone to the disorder, and a phthisical subject becoming pregnant will often improve until after her confinement, when an exacerbation of the disease will occur, which has usually a fatal result. (4) Unhealthy surroundings play a most important part in the production of phthisis, and indoor occupations such as those of lacemakers and city clerks are specially unfavourable. A damp soil undoubtedly favours the production of the disease—even the dampness from faulty construction of a dwelling will do so. A moist, hot atmosphere, such as exists in

TABLE VIII.—THREE STAGES OF PHTHISIS.

Anatomy. (See Fig. 46, p. 178.)	Physical Signs.	Symptoms.
(a) CONGESTION of lung tissue, consequent on invasion by tubercle bacilli.	At apex of the lung— (i.) Feeble R.M., with occasional fine crepitation heard at end of inspiration ; or (ii.) Unduly harsh breathing with a prolonged expiration.	(i.) Increasing languor on exertion ; (ii.) Slight morning cough ; (iii.) Slight rise of temperature ; (iv.) In some cases hæmoptysis. (v.) Anæmia in some cases.
(b) CONSOLIDATION— due to the hyperplasia, cell infiltration, and the fusing together of the tuberculous foci.	Over diseased part, usually at apex, are : (i.) Impaired movement ; (ii.) Flattening ; (iii.) Increased vocal fremitus ; (iv.) Dull percussion note ; (v.) Bronchial or tubular breathing ; (vi.) B r o n c h o p h o n y (increased V.R.).	(i.) Weakness and emaciation increase ; (ii.) Temperature markedly higher in the evening ; (iii.) Night sweats ; (iv.) Anæmia.
(c) BREAKING DOWN and EXCAVATION.	Signs as in (b), plus— 1. Moist clicking râles ; and later on— 2. Signs of presence of cavity : (i.) C a v e r n o u s breathing. (ii.) Post-tussic suction ; (iii.) Pectoriloquy ; (iv.) Râles with metallic tinkle.	All the above symptoms aggravated : (i.) Cough distressing, with quantities of nummular e x p e c t o r a t i o n ; h æ m o p t y s i s may be profuse ; (ii.) Temperature high, and with wide range ; (iii.) Sometimes diarrhœa etc.

certain factories, favours the spread of the disease. A dust-laden atmosphere, such as that of stonemasons, knife-grinders, tin and copper miners, fustian-cutters, is a potent cause of phthisis. (5) The 1911 report of the Royal Commission on Tuberculosis confirms the view that tuberculosis in mankind is due to two types of tubercle bacillus, one of human and one of bovine origin. Pulmonary tuberculosis is usually due to infection by the human bacillus, which is conveyed by air tainted with dried sputum containing living bacilli—hence the importance of destruction of the sputum. In children, on the other hand, the bacillus is usually of bovine origin, and it is found chiefly in the abdomen (peritoneum or glands), bones, joints, cervical glands, and in the lungs when acute miliary tuberculosis carries the bacillus from an infected focus to the blood-stream. Recent work by Dr. A. P. Mitchell proved the connection between

unsterilised milk containing bovine tubercle bacilli and enlarged cervical glands in the children around Edinburgh. The bovine bacillus enters the body *via* the alimentary canal and tonsils, and it has been proved that the disease is due to the ingestion of infected milk of tuberculous animals. It is, however, an undoubted fact that mankind is naturally resistant to the tubercle bacillus. Birch-Hirschfield undertook a laborious investigation of 4,000 post-mortems, and he found that in 40 per cent. of these persons, dying from all manner of diseases, the lungs showed evidences of tubercle which had undergone spontaneous recovery. In view of these facts, and that most of the predisposing causes above mentioned are preventable, there is no reason why phthisis should not one day become as rare in England as leprosy is to-day.

Prognosis.—1. Usual *course* and *duration*. Phthisis is essentially a chronic but progressive disorder, and until recently nearly all cases applying for treatment terminated fatally. The death-rate from phthisis in 1838 was 38 per 1,000 ; in 1892, 14 per 1,000 living ; and in 1910 13·4 per 1,000 living. Rapid cases may terminate in death in the course of three to six months. When the disease is indolent, and the patient resistant to the microbe, it may drag on for years. There are four chief modes of death, which in order of frequency are—(1) asthenia, (2) hæmoptysis, (3) asphyxia from pneumothorax, (4) the occurrence of other complications.

2. The prognosis in reference to *Causation* depends on : (i.) Heredity. Unquestionably it takes a more favourable course and the process tends to be less active in cases where there is no family history of tuberculosis. (ii.) The age of the patient influences the course considerably, for it is much more rapid in the young than in people over thirty. (iii.) The hygienic surroundings of a patient, as we shall see under *Treatment*, make considerable difference to the course of the disease. Where the patient is well-to-do and can be removed from those conditions which have promoted the disease, he has a good 'chance nowadays of recovery ; but among the poor, who are forced to continue among their squalid surroundings and at their work, a fatal issue almost necessarily results. (iv.) Previous alcoholic excess diminishes the chance of recovery.

3. *Untoward Symptoms.*—(i.) Undoubtedly the most important feature is the temperature. Not only is an active tuberculosis evidenced by pyrexia, but the degree of fever, and still more the extent of the variations, are a fairly precise measure of the activity of the tuberculous process. (ii.) The condition of the lung is of course important. The presence of râles, as denoting softening and advancing disease, is unfavourable, and their disappearance favourable, but the extent of lung involved is as important a factor in prognosis. Thus, a man in the third stage, with a cavity at the apex in one lung, and little disease elsewhere, has a better chance of recovery than one with slight tuberculous foci scattered through the lung. If both lungs show disease in the third stage, recovery is rare, though health has been restored in some cases after prolonged treatment.

(iii.) The general symptoms also aid us in recognising the rate of progress. When the weight is increasing, the temperature declining, and food is taken well, and when anæmia and muscular weakness are no longer marked, the chances of recovery are good.   (iv.) Early hæmoptysis does not affect the prognosis in any way, but occurring later in any quantity is apt to weaken the patient considerably.

4. *Complications.*—The presence of complications is undoubtedly bad. The commonest complications are : (1) Pleurisy, which is very frequent, but is often of a conservative nature, for adhesions may sometimes prévent pneumothorax ; (2) tubercle may occur in other parts—the peritoneum, meninges, and especially in the intestine, giving rise to ulceration and an exhausting diarrhœa [1] ; (3) the larynx may be affected either previously or subsequently, and undoubtedly it adversely influences the prognosis ; (4) lardaceous disease of the liver, spleen, and other organs used to be frequently seen ; (5) pneumothorax and 'pyopneumothorax may ensue from the bursting of a cavity into the pleura—fatal asphyxia *may* result (§ 104) ; (6) thrombosis of various veins is a less common complication ; (7) peripheral neuritis is now a recognised occurrence, sometimes very early in the disease ; (8) vomiting.

It is a good rule never to commit yourself to an opinion on any case of phthisis without first noting the effects of treatment.

Treatment of Phthisis.—The subject of treatment will be dealt with under four headings : (*a*) remedial ; (*b*) symptomatic treatment ; (*c*) treatment by tuberculin ; (*d*) the open-air treatment ; and (*e*) preventive measures.   The indications of all treatment are to reduce the inflammation, to destroy the virus, to build up the strength, and to palliate the symptoms.

(*a*) The *Remedial Treatment* formerly in vogue was mainly directed to building up the strength by means of cod-liver oil, maltine, hypophosphites, and other tonics.   Cod-liver oil is of great value in treating afebrile cases.   Guaiacol, 20 grains (1·3), gradually increased, creosote, eucalyptus and other antiseptics may be given at any time.   Thiocol, gr. v. (0·3), thrice daily has all the advantages, without the drawbacks, of creosote. By some these are also administered as an injection into the lung.   Perhaps the best kind of treatment on this line is the use of a spray four times a day, lasting fifteen minutes, of formalin.[2]   Inhalation of antiseptics may be administered by Yeo's respirator.   Counter-irritants were largely used to reduce the inflammation, the favourites being iodine or croton oil applied over the apex of the lung.   Where possible these patients. should live in the mountains, where the dry and rarefied air compels deep breathing and the dust of cities is avoided.   A liberal diet is necessary.   Abdominal breathing is recommended, so that the apices may be

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[1] Diarrhœa may also occur as part of the hectic fever without any ulceration of the bowels.

[2] The following formulæ are recommended (Formalin = 40 per cent. of Formic Aldehyde) : Formalin, ℥i. (4) ; Glycerine, ℥iv. (16) ; Aq. Dest. ℥v. (160) (Dr. Lardner Green).   Formalin ℳ x. (0·6); menthol, gr. ѵi (0·3) ; Cocain (alkd.), gr. v. (0·3) ; Paraff. liq. ad ℥i (32) (Dr. V. E. Sorapure).

at rest. *Artificial Pneumothorax* offers sometimes excellent results in cases which were previously considered hopeless. In every case of unilaterally active pulmonary tubercle the progress of which is not arrested by ordinary sanatorium treatment, this method should be tried. Success or failure depends largely on the extent of pleural adhesions present. Artificial pneumothorax is specially indicated when there is cavity formation ; not only in tubercle, but also in pulmonary abscess, and in some cases of bronchiectasis. In severe hæmoptysis the results are striking. Regular refills of air are required ; the lung must be kept collapsed, and the treatment prolonged on an average for three years or more. Work may be usually resumed early in the treatment— a decided economic advantage. The sputum commonly becomes free from bacilli after a few months and therefore the patient is no longer infectious. The drawback is that this method requires special training and constant control by X-rays, and must therefore be left to the expert.

(b) *Symptomatic Treatment.*—It will be seen that in the third stage there is not much hope of recovery, but even in the worst cases we can ameliorate the symptoms, and so ease the passage to the grave. (1) For the cough, tinct. camph. co. and other expectorants are not of much use. The best cough mixture is one containing liquor morphine, or, better still, codeia in small doses with dilute sulphuric acid. Warm alkaline drinks promote expectoration. (2) Night sweats, which are often very profuse and exhausting, may be combated by atropine, zinc oxide, picrotoxin, and strychnine, especially the first named. Night sweats are said to be seldom troublesome if there be free exposure to fresh air. (3) The diarrhœa is also very exhausting, and must be combated with catechu, opium, intestinal disinfectants, and mineral acids. (4) Pleuritic pains may be eased by stupes, or painting with tincture of iodine. (5) The concurrent dyspepsia must be combated in the usual way, but the vomiting is often a very troublesome symptom, and there are three kinds of vomiting which admit of three different methods of treatment. (a) If preceded by nausea, it points to disorder of the stomach, and should be treated by bismuth, etc., on the usual lines. (b) If the vomiting be preceded and caused by coughing, it is a good plan to give hot drinks just before a meal, in order to encourage the expectoration and get the paroxysms of coughing over before the meal is commenced. (c) If neither of these causes can be traced, the vomiting is probably due to irritation of the vagus, and may sometimes be relieved by opium. Sometimes vomiting is controlled by the will. (6) The treatment of hæmoptysis, pneumothorax, and laryngeal ulceration are dealt with elsewhere.

(c) *Treatment by Tuberculin* depends upon the principle of immunisation (§ 416). Koch followed his discovery of the tubercle bacillus by soon afterwards issuing to the world the toxin produced by the bacillus. This he called tuberculin, and it is now called *old tuberculin.* It was administered hypodermically and produced considerable " reaction "—*i.e.*, constitutional disturbance—in the patient. It was largely used, but the

results were not satisfactory. There are now several tuberculins on the market. The initial hypodermic dose (1/500,000–1/50,000 mg. TR. or BE.) is gradually increased until the patient can tolerate larger doses without any rise of temperature or excessive local reaction ; in febrile cases the initial dose must be extremely small and the increase in dosage very gradual. If either local or general reaction occurs, the dose is diminished and the interval between the doses is increased. Tuberculin should not be administered except by those who are conversant with the methods of controlling dosage of vaccines. Others report good results from larger doses.

(*d*) The " *open-air*," hygienic, or sanatorium treatment of phthisis, as it is now called, is not altogether a new method, for fresh air has always been advocated as advantageous to these patients. Systematic open-air treatment was first established at Nordrach. There are now numerous sanatoria both at home and abroad. Much discussion has taken place as to whether the treatment cannot be carried out without a sanatorium. Among the well-to-do, perhaps, a sanatorium is not indispensable, but in the middle and lower classes the necessary discipline cannot be otherwise carried out. That residence in a sanatorium is not absolutely necessary is evidenced by cases which have been under my care, even in an advanced stage of phthisis, who were unable to go away. One of them spent all the daytime in Kensington Gardens, in all weathers, and when indoors the windows were always open. The patient recovered in six months.[1]

Briefly, the *advantages* gained by this method of treatment consist of : (i.) Increased medical supervision from day to day and hour to hour by the medical officer of the sanatorium ; (ii.) the continuous exposure of the patient to fresh, pure air, night and day, the windows never being shut and sometimes wholly removed ; (iii.) systematic exercise in suitable cases ; (iv.) the ingestion of nourishing and sufficient food ; (v.) a suitable amount of rest during the fever stage, and a freedom from excitement ; (vi.) the avoidance of mixed infections by hygienic mode of life. Cleanliness and fresh air tend to obviate pyogenic processes and infections. All possibility of the introduction of influenza and other infective disorders should be avoided by the *proper regulation of visitors* to patients. I believe that some day these latter will be subjected to the most rigorous scrutiny and inquiry before being allowed to come in contact with the consumptive patients in a sanatorium. The mixed or superadded infections do more harm than the tubercle bacillus. This is probably the reason why tuber-·culous patients do so badly in the wards of a general hospital. The treatment varies at the different sanatoria. In some, graduated labour is the chief feature ; in some the high altitude ; in others tuberculin injections form important factors in the treatment. Patients who return to ordinary life, return with a working knowledge of the hygienic rules appropriate for consumptive subjects.

The possible *disadvantages* urged are : (i.) The fear of hyper-medication

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[1] See also a case reported in the *Lancet*, January 20, 1900.

that may go on in sanatoria ; and (ii.) certain unsuitable cases (see below) may be deleteriously affected.

In carrying out sanatorium treatment, seven rules should be observed :

(1) Much depends on the suitability of the case, and the *earlier the stage* the better. There are three conditions in which the sanatorium, or open-air treatment, is undesirable : (i.) When the process is too active, as evidenced by a high and wide range of temperature ; [1] (ii.) when the lungs are too far destroyed ; and (iii.) when the case is attended by active bronchial catarrh.

(2) The food must be abundant, and the cuisine appetising and attractive.[2] But here an important caution comes in, else the patient puts on fat without influencing the disease. The food must be in proportion to the exercise, and the patient's weight should never much exceed his previously normal weight. The proteid food should be increased relatively to the farinaceous, otherwise the patient becomes plethoric and breathless.

(3) Evidences of benefit should be carefully looked for. They are three in number : (i.) A lowering of the temperature and a lessening of its range ; (ii.) an increase in the appetite ; (iii.) increase of weight *combined* with the two previous features.

(4) In deciding the important question of rest or exercise, the great value of accurate temperature records is again seen. The system of graduated labour introduced at Frimley Sanatorium by Dr. Paterson[3] is being followed by many with excellent results. There are six grades of labour, varying from walking exercise, carrying heavy implements, to the full work of a navvy. The patient is not allowed to begin work until the temperature is stable at not over 99° F. in males, and 99·6" F. in females. If it rises after slight exercise, the patient rests until it is normal. Progressively heavier work can be performed without any rise of temperature. The normal and mental effects are invigorating and enable the patient to resume ordinary occupation after leaving the sanatorium with a healthier standpoint than after a long rest with idleness.

(5) Amusement is necessary, but it requires to be carefully regulated. The patient should not talk too much, and any excitement or heated ·discussion is bad. *The whole day, and, if possible, the night also, should be spent out of doors, no matter what the weather may be*, and outdoor amusement cultivated. A very useful contrivance is a small revolving summer-

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[1] Some do not regard this as a contra-indication to open-air treatment ; but in such cases the length of the journey has to be considered, and the undesirability of mixing such patients with others.

[2] Thirteen pints of milk, or its equivalent, is in most institutions adopted as a fair standard of diet ; that is, 9 ounces of proteid, 7¾ ounces of fat, 10¼ ounces of carbohydrate : total, 27¼ ounces water-free food. With " Parkes' Hygiene " percentage composition tables, varying diets can easily be made up containing the above proportion of food elements.

[3] " Auto-inoculation in Pulmonary Tuberculosis," by Marcus Paterson, 1911. The continual auto-inoculation induced by exercise sets in motion the protective mechanism of the blood.

house, the front of which is open, and can be turned away from the wind.

(6) The duration of the treatment must be sufficient, and should be continued for some time after all symptoms have disappeared. If the case is only in the first stage, cure may be accomplished in six months.

(7) The hygiene and the locality of the building are important matters, but the reader must refer to special works for this. The beneficial effect of mountain air has been proved beyond dispute. It is particularly indicated in cases of consolidation without cavitation, but later stages also can be benefited. Deeper and longer respirations are taken at high altitudes, the air is purer than elsewhere, and the red blood cells are increased.

(e) *Preventive Treatment.*-- Since the microbic origin of tuberculosis was admitted, the question of how far it is a contagious disease has been keenly debated. The results of the labours of the Royal Commission are mentioned above (p. 182), and from these conclusions it is obvious that preventive measures come under the headings of (1) prevention of the communication of the disease from man to man; (2) prevention of its extension from animals to man; and (3) education of the public. (1) Prevention of infection from man to man is ensured by destruction of the microbe, and by strengthening the resisting powers of those hereditarily predisposed to the disease. For the destruction of the microbe the rooms in which the phthisical people have lived must be thoroughly disinfected; and the sputum must be destroyed before it dries. The patient must spit only into some portable receptacle containing a disinfectant such as lysol, or into paper sputum cups which can be burned. Tuberculous patients should not share the sleeping rooms of healthy individuals. (2) The method of prevention of infection from animals is a matter for the consideration of the State. Bovine tubercle is conveyed by the ingestion of the flesh or products of diseased cattle. In order to protect the community from this danger it is necessary to have adequate inspection and full powers of dealing with infected meat and milk. (3) There are many ways of educating the public on the hygiene of the home in tuberculous families. Tuberculosis exhibitions, lectures, etc., merit the support of every medical man. The most practical scheme at present, however, is the multiplication of tuberculin dispensaries. Sanatoria, hospitals, and dispensaries ought to work in association. At the dispensaries the early cases of tuberculosis are detected and drafted off · to sanatoria; the homes of the invalids are visited and the inmates are instructed as to the correct hygienic measures to adopt in order to prevent contamination of those uninfected persons who have been in contact with the disease. Treatment is also given at the dispensaries, but their greatest sphere of usefulness lies in their educative influence.

§ 111. **Fibroid Phthisis** is one of the least common of the varieties of pulmonary tuberculosis. It may be defined as a tuberculo-fibroid disease of the lungs, occurring for the most part in elderly subjects, running a protracted course, and terminating

in contraction of the lung. This disease is very apt to be confused with chronic interstitial pneumonia or cirrhosis of the lung (§ 114).[1]

Symptoms.—The disease is essentially one of insidious onset and long duration. The patient complains of a chronic cough for many years. Later on this may become paroxysmal, and especially troublesome in the morning. Progressive shortness of breath, clubbed fingers, slowly increasing weakness and emaciation, with little or no fever, constitute the other symptoms.

The *Physical Signs* begin and are almost always most marked at the apex. *Both lungs* are usually affected (which contrasts with interstitial pneumonia), but the signs of disease are afterwards more advanced on one side. There is impairment of the chest movement, and later on contraction of one side of the chest. The area of præcordial dulness is increased when the left lung is involved ; and the heart and other viscera may be displaced. The signs of consolidation, with gradual softening, may also be present. Hæmoptysis sometimes occurs, and the tubercle bacillus may be discovered on careful and repeated examination of the sputum.

The *Diagnosis* from other forms of *phthisis* is made by the extremely protracted course of this disease and the age of the patient. *Chronic interstitial pneumonia* resembles it very closely, both in its physical signs and symptoms, and the diagnosis of interstitial pneumonia can only be inferred (i.) from the absence of the tubercle bacillus after oft-repeated examinations, and (ii.) from the more usual localisation in one lung.

Etiology.—Fibroid phthisis is more frequently met with at and after middle life. It may follow chronic bronchitis, broncho-pneumonia, or repeated attacks of pleurisy. In true Fibroid Phthisis the tubercle bacillus primarily deposited in a healthy lung under the same circumstances as in chronic pulmonary tuberculosis, and then causes an indolent fibroid reaction. On the other hand, chronic interstitial pneumonia may become the seat of tuberculous invasion, and in that case the causes of chronic interstitial pneumonia are the causes of fibroid phthisis (see Interstitial Pneumonia, § 114).

Prognosis.—Its course is very indefinite and protracted. Sometimes acute tuberculosis supervenes. The chief complications are bronchiectasis, compensatory emphysema of the lungs, lardaceous disease of other organs, and cardiac failure. In general terms the prognosis depends upon the same conditions as those of pulmonary tuberculosis and the *Treatment* is the same.

II. *The patient complains of breathlessness ; on examining the chest,* **dulness** *is found at one or both* **bases,** *and* SIGNS OF FLUID *are detected there. The disease is* HYDROTHORAX.

§ 112. **Hydrothorax** is a chronic collection of serous fluid in the pleural cavity, differing from the effusion of pleurisy in being non-inflammatory.

Symptoms.—The general symptoms may be but little marked if the fluid is small in quantity. The onset is usually gradual. Dyspnœa is generally present, especially on exercise, but its degree depends upon the amount of fluid. As hydrothorax is always a secondary condition, the symptoms may be masked by the presence of dropsy elsewhere ; and it is remarkable how often hydrothorax is overlooked on this account. In rare cases the fluid collects with great rapidity.

The *Physical Signs* are those of fluid in the chest (*vide* § 89). The level of the fluid in hydrothorax, unless excessive in quantity, moves when the patient alters his position, thus differing from the inflammatory fluid of acute pleurisy. This is an important diagnostic feature which can always be elicited, except when the fluid is confined by adhesions.

Diagnosis.—The disease has to be diagnosed from other disorders giving rise to dulness on percussion (§ 106). As regards *pleurisy,* in addition to the mobility of the

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[1] Reference to chronic interstitial pneumonia (§ 114) will show to what condition the term " fibroid phthisis " should be confined.

fluid, hydrothorax is distinguished by the absence of pyrexia at the onset, by the absence of pain, and by the fact that the fluid occurs usually on both sides.

Etiology.—(i.) Hydrothorax may form part of the *general* dropsy of Bright's disease, in which circumstances both pleuræ are involved. Here the hydrothorax is of no very great importance *per se*, but the onset of dyspnœa in Bright's disease should always direct our attention to the pleuræ. (ii.) Similarly, it may form part of *cardiac* dropsy, in which circumstances one pleura (the right) is often solely or chiefly affected. (iii.) New growths in the chest are generally attended by hydrothorax. This is especially so in the case of cancer, which should always be suspected in the aged. In this case the fluid is blood-stained, and may be found to contain cancer or sarcoma, cells. In tubercle there is rarely much fluid in the pleura, adhesions being more common. (iv.) Aneurysm or other intrathoracic tumours pressing on the veins of the thorax may give rise to hydrothorax on one or both sides.

Prognosis.—The disease is essentially chronic, the duration depending very much upon the cause. In general terms the prognosis of the condition is unfavourable. The patient should be carefully watched for the occurrence of shivering, sweating, or intermitting pyrexia, as indicative of empyema. The sudden onset of signs of fluid in the chest, accompanied by shock or collapse, in a case which has previously presented the symptoms of aneurysm, points to the occurrence of hæmorrhage into the pleural cavity (hæmothorax).

Treatment.—The treatment is comparatively simple. The administration of brisk hydragogue purgatives will generally reduce the amount of fluid ; if this fails, or if the fluid return, or in any case where dyspnœa is extreme, paracentesis (§ 98) should be resorted to. The operation of tapping may be repeated indefinitely. Diuretics or cardiac stimulants are useful. For the rest, the treatment must be directed to the primary condition (see also § 98).

III. *The patient complains of breathlessness ; on examining the chest,* dulness, *usually slight, is found at one or both* bases, *and on auscultation,* FINE CREPITATIONS *are heard. The disease is* PULMONARY CONGESTION OR ŒDEMA.

§ 113. **Œdema of the Lung** (Pulmonary Congestion).—Œdema of the lung is a serous exudation into and around the air vesicles. It is synonymous with the term "hypostatic congestion," or, as it is sometimes called, "hypostatic pneumonia." It determines the end of many serious disorders.

Symptoms.—(i.) It is never a primary condition, and therefore our attention is first directed to the symptoms of its cause. The advent of hypostatic congestion is always insidious, and it is only by careful watching that it can be detected. (ii.) A considerable amount of dyspnœa is present, which may amount to orthopnœa. (iii.) There is a frothy mucous expectoration, not infrequently tinged with blood.

The *Physical Signs* are somewhat indefinite but they are found, as is implied by the term "hypostatic," chiefly at the bases of both lungs. The percussion note is somewhat impaired, and the air entry at the bases is diminished, and is attended by abundant moist crepitations.

Diagnosis.—The condition is diagnosed from true pneumonia by the gradual onset, the indefinite signs, and the absence, for the most part, of pyrexia, and other constitutional symptoms. Any rise of temperature that may be present is due to the primary or causal condition.

Etiology.—(i.) The disease is most frequently met with in elderly people. (ii.) Pulmonary œdema complicates various blood disorders and fevers, especially typhus and typhoid fevers. The latter, indeed, is so frequently complicated in this way that hypostatic congestion is an aid to the diagnosis in the second and third weeks of the disease. In Bright's disease and anæmia, œdema of the lungs occurs as part of a generalised dropsy. (iii.) Cardiac and other diseases, leading to mechanical dropsy, produce œdema of the lungs. (iv.) Tumours pressing on the veins within the mediastinum may result in pulmonary œdema.

Prognosis.—The prognosis is always grave, because pulmonary œdema indicates

either considerable impediment to the circulation in the lungs, or a serious toxic condition of the blood. It frequently terminates life in circulatory disorders, and in specific fevers of the asthenic type. In pneumonia it heralds a fatal issue. The extent of the œdema is indicated very fairly by the degree of dyspnœa.

Treatment.—The indications are to relieve the cause, if possible, and to stimulate the heart. Ammonium carbonate and other stimulating expectorants aid the heart and promote expectoration. The liberal administration of alcohol and other diffusible stimulants is called for. In the aged, among whom even slight disorders are apt to be attended by pulmonary œdema, it is well *to keep the patient propped up* in a semi-recumbent posture. For the same reason it is advisable, in cases of fracture and other surgical maladies in the aged, to get them up as soon as possible, even at the risk of doing harm to their surgical ailment, so as to obviate the occurrence of hypostatic congestion of the lungs.

GROUP B.—*We now turn to the* **rarer** *chronic diseases attended by* **dulness** *on percussion, in which the dulness occurs in irregular and* **scattered areas** : IV. INTERSTITIAL •PNEUMONIA ; V. THICKENED PLEURA ; VI. CANCER AND OTHER TUMOURS ; VII. COLLAPSE ; VIII. SYPHILITIC DISEASE ; and IX. MEDIASTINAL TUMOURS.

§ 114. Chronic Interstitial Pneumonia--apart from that form due to the malign effects of certain trades—is a rare disease. It may be defined as a chronic interstitial fibrosis of the lung, localised or diffuse, according to the variety, running a protracted course, and resulting in contraction of the pulmonary tissue.

An increase of the fibrous tissue of the lung may take place under the following conditions, all being chronic processes :

(i.) An indolent tuberculous process may assume a fibroid character. Fibrosis is one of the ordinary terminations of a tuberculous focus ; but when the progress is very slow and protracted, with excessive formation of fibrous tissue, it constitutes true *fibroid phthisis.*

(ii.) The constant inhalation of dust in certain trades (*e.g.*, fustian cutters, jute workers, wool-sorters, stone, knife, and other grinders and polishers, iron and coal miners, etc.) gives rise to a *chronic broncho-pneumonia,* followed by a peribronchial fibrosis, which later on involves considerable areas of lung tissue.

(iii.) Repeated attacks of *pleurisy* may be attended by a subpleural fibrosis (thickened pleura), and dense bands of fibrous tissue may extend into the lung (Sir Andrew Clark).

(iv.) *Acute broncho-pneumonia,* becoming chronic, may, although very rarely, result in an interstitial fibrosis. This form very often terminates by becoming tuberculous.

(v.) An *acute lobar pneumonia,* similarly, may assume a chronic course, and may result in an interstitial fibrosis (Addison). This form has not the same tendency to become tuberculous.

(vi.) *Syphilitic disease* of the lung is rare, except as a congenital manifestation in infancy, in which circumstances the change consists of a fibroid induration of the lung (Kingston Fowler).

All these may become the seat of tuberculous disease, but only the first, which is a *tuberculo-fibroid* process, should be called " fibroid phthisis." The other varieties constitute cirrhosis of the lung, and if they are invaded by the tubercle bacillus, they form a *fibro-tuberculous* process, which in its later stages may be indistinguishable from fibroid phthisis.

The general *Symptoms* consist of progressive weakness and dyspnœa. There is no fever unless there is ulceration of the bronchi or septicæmia—a common occurrence in late stages of the disease.

The *Physical Signs* may be found either at the base or the apex, though usually the former. Except in the variety due to the inhalation of irritating particles, only

one lung is involved, thus differing from fibroid phthisis, in which both lungs are usually affected. There is deficient mobility of the diseased side, which later on undergoes contraction, so that there may be considerable difference in the measurement of the two sides of the chest. There is dulness on percussion. On auscultation, bubbling râles may be heard, but sometimes the only symptom is weak bronchial breathing or a weak respiratory murmur. The expectoration sometimes contains blood, but never the tubercle bacillus.

The *Diagnosis* of interstitial pneumonia from fibroid phthisis is sometimes very difficult, as may be seen in the description of the various processes just named.   It is also liable to be mistaken for empyema. X-ray photographs may afford very great assistance.

The *Etiology* of the condition is given above. It is met with chiefly in male subjects under the age of fifty—especially between fifteen and thirty. Alcoholism predisposes. The commonest form of chronic interstitial pneumonia -is that met with in persons engaged in trades attended by the inhalation of irritating particles. Sometimes it is a sequel to other pulmonary disorders.

Prognosis.—The prognosis is serious, because nothing will remove the fibrous tissue. As regards the duration of life, the prognosis is good'if the patient is not losing weight and the disease is not too extensive. The *complications* are bronchiectasis, a very frequent sequel, dilated right heart, and emphysema occurring in other parts of the lung. When ulceration of the bronchi has taken place, lardaceous disease and septicæmia may ensue.

Treatment.- Counter-irritation and respiratory exercises are given on the lines advised in Chronic Phthisis (§ 110). Formula 61 is useful.

§ 115. V. Thickened Pleura is a condition which sometimes succeeds dry pleurisy, especially recurrent dry pleurisy. It is important to be able to recognise it, lest it should be mistaken for some more serious condition, though it is somewhat difficult to diagnose. It is more often localised to one part, and that most commonly at the apex associated with chronic phthisis. The *symptoms* are : (i.) A localised enfeeblement of the respiratory murmur ; (ii.) dulness on percussion ; and (iii.) diminution in the vocal resonance and fremitus.

The *diagnosis* is arrived at (i.) by the history of the case—*e.g.*, there has been an attack of pleurisy or pneumonia in the past—and (ii.) by the absence of signs of active disease when the patient is kept for some time under observation. The condition is often discovered only by chance, when the patient seeks advice for other ailments. Treatment is of no avail ; and, if only moderate in degree, the disease is not of much consequence. Counter-irritation may be applied.   .

§ 116. VI. Malignant Disease of the Lung.—Cancer of the lung is rarely a primary condition, but is most frequently secondary to cancer of the · breast or abdominal organs. The most common form of malignant disease, sarcoma of the mediastinum, is described in § 66. A primary malignant growth tends to involve one lung : secondary growths tend to be disseminated in both lungs. The evidences of the former are usually more distinct than those of the latter.   ●

Symptoms.—The lung trouble may be preceded by signs of malignant disease elsewhere. The first evidence of involvement of the lung is breathlessness, followed by cough and by expectoration, which may from time to time be tinged with blood (" prune-juice sputum "). Pain is often present, and indicates generally that the pleura is invaded, in which case there is usually a certain amount of pleuritic (blood-stained) effusion.

The *Physical Signs* are often very indefinite. Clinically, there are two forms— (a) The *nodular* form is usually attended by serous effusion (see Hydrothorax, § 112). Effusion into the pleura coming on slowly, or returning persistently, in an elderly person not the subject of phthisis is of itself suspicious, and the diagnosis is confirmed when, on aspiration, the fluid is found to be blood-stained. Sometimes in the midst of what appears to be a hydrothorax we detect the signs of consolidation ;

indicating that the neoplasm has come to the surface in one locality.   Enlarged veins are often seen on the chest wall.

(b) With the *infiltrated* form we find signs of consolidation, accompanied later on by the moist sounds due to the breaking down of the growth.   Here again nearly every variety of physical sign may be met with in different parts of the lung, and if the main bronchus be obstructed, there is entire absence of the breath sounds.

Diagnosis.— The condition has to be diagnosed from different forms of pneumonia, from pleurisy with effusion, and from hydrothorax.   The type of cell found in the effusion after paracentesis is diagnostic in many cases.   The age of the patient, the course of the affection, the absence (usually) of much pyrexia, the presence of enlarged glands and cachexia should enable us to come to a conclusion.   The X-ray picture may be conclusive, especially when taken after removal of fluid.

Prognosis.—The question is one of duration, and this can only be gauged by daily observation of the case, and by noting the rate at which the growth appears to be spreading.   Death usually occurs in about six months.

The *Treatment* resolves itself into the relief of pain and the amelioration of other symptoms.   X-rays in large doses give great temporary benefit.

§ 117. Hydatid Cyst is a more frequent disease of the pleura or of the lung in Australia than in this country.   No symptoms may be experienced by the patients for a long time.   If superficially situated, it causes bulging on the chest wall.   The physical signs resemble those of pleural effusion, but the dulness has a more rounded outline.   When a cyst occurs at the apex of a lung, it is usually mistaken for tubercle.

The *diagnosis* may be impossible from examination of the chest, until an X-ray examination is made.   Eosinophilia is nearly always present, and aids the diagnosis.   The characteristic hooklets may be expectorated, and hydatids may be present in other organs.   The serum of the patient gives a specific precipitin reaction.

Prognosis.—The cyst may rupture into the pleura or into the lung, and cause haemoptysis or abscess.   Serious constitutional symptoms may arise from the onset of suppuration.   Or it may open into a bronchus, thus leading to spontaneous recovery.

The *treatment* is mainly surgical.   Aspiration should be avoided.

§ 118. VII. Atelectasis, or Collapse of the Lung, is a condition in which the lung tissue is in an unexpanded state.   The term " atelectasis " is usually applied to lung tissue which has never properly expanded, a congenital condition, due to imperfect development.   The term " collapse of the lung " is applied to lung tissue which has previously expanded, but in which the air vesicles have subsequently collapsed.

· Atelectasis is a *congenital* condition, of which symptoms occur in the new-born child, and consist of cyanosis, with shallow, rapid respiration.   The lower part of the chest is drawn in by each respiration.   On auscultation, the respiratory murmur is found to be very faint.

The *Symptoms* of collapse of the lung follow and complicate those of the disease which has led to the condition ; for instance, the patient may not recover so rapidly as he ought, or the breathing is more embarrassed than can be accounted for by the concurrent disease in the chest.   The physical signs vary considerably with the degree of collapse.   Thus :

(a) In *complete* collapse of a part of the lung, as, for instance, in collapse due to compression or complete obstruction of a bronchus high up, there is impairment of the percussion note, with a diminution or absence of the breath sounds and of the vocal resonance and fremitus.

(b) Where the collapse is only *partial* in degree—e.g., where the bronchi remain patent, as occurs sometimes when the lung is compressed by pleuritic or pericardial effusion—there are signs resembling those of consolidation (§ 89), except that the percussion dulness is not so marked, and the breath sounds, though bronchial in character, are somewhat feeble.

(c) Where the collapse is *slight* and limited, the chief sign is an enfeebled respiratory

murmur. In addition, during deep inspiration are heard fine rustling crepitations, due to the expansion of the collapsed vesicles.

The *Diagnosis* is made usually by the existence of a causal condition. When this is detected, attention may then be directed to the physical signs of the lungs. It will be observed that the signs of partial collapse resemble the signs of consolidation, and those due to slight collapse resemble early pneumonia.

Etiology—The causes are of four kinds : (*a*) Causes which produce *obstruction*, such as (i.) a tumour at the root of the lung (*e.g.*, aneurysm); (ii.) obstruction in the throat (*e.g.*, adenoids); (iii.) stricture of a bronchus (*e.g.*, gumma); (iv.) secretion obstructing the bronchi, though this is only sufficient to cause obstruction in children suffering, for example, from whooping-cough, or broncho-pneumonia; (v.) foreign bodies obstructing the larynx or bronchus. (*b*) *Compression* of the lung may be produced by pleural or pericardial effusion, an enlarged heart, or tumours of the mediastinum, or of the abdomen. The condition is often the result of spinal curvature. It may occur after abdominal operations and anæsthesia, and give rise to difficulty in diagnosis, unless the possibility of its occurrence is borne in mind. (*c*) *Paralysis* of the intercostal muscles or diaphragm, as in diphtheria or other cause of neuritis. (*d*) Injury to the chest wall with or without involvement of the thoracic contents, especially by high velocity projectiles, is a fertile cause of collapse of lung either homolateral or contralateral.

In *adults* collapse is most often met with as the result of pleural effusion or tumours in the chest; in *children*, of bronchitis or broncho-pneumonia.

Prognosis.—The course of the disease depends very much upon the cause. Recovery as a rule soon takes place after compression by effusions, obstruction or stricture of the bronchi, and throat affections.

The *Treatment* is unsatisfactory. It should be directed to the removal of the cause, and especially to the promotion of recovery of any concurrent pulmonary disorder. That form which yields best to treatment is met with in children with bronchitis and broncho-pneumonia. In adults it might be well to try the efficacy of respiratory exercises.[1]   •

§ 119. VIII. Syphilis of the Lung.—In infants this disease may take one of two forms : (*a*) The pneumonic condition of lung, which is found in infants, usually still-born, is universally regarded as an interstitial pneumonia of syphilitic origin. (*b*) Gummata are occasionally met with in the lungs of infants who are the subjects of hereditary syphilis; still more rarely they are met with in adults. Dyspnœa is usually the only symptom. The signs are those of consolidation, and collapse. In adults syphilis of the lungs may take other forms—*e.g.*, broncho-pneumonia, bronchiectasis, etc.—and may lead to extensive infiltration and breaking down, or to fibrosis.

GROUP C.—CHRONIC DISEASES attended by **Hyper-resonance** on percussion : I. In quite nine out of ten cases of hyper-resonance it exists on both sides, and is due to EMPHYSEMA. There are five other conditions which give rise to it—namely : II. PNEUMOTHORAX (§ 104) ; III. SKODAIC RESONANCE (§ 87) ; IV. A very large CAVITY in the lung (Phthisis, § 110) ; V. A TUMOUR between the chest wall, and a large bronchus (§ 116) ; VI. • DISSEMINATION OF SOLID MATERIAL through the lung in certain exceptional circumstances (*e.g.*, pneumonia, sarcoma, etc.). The diagnosis of these various conditions is given in the form of a table (p. 196). All except EMPHYSEMA are described elsewhere.   •

I. *The patient has complained of breathlessness for some years. There is*

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[1] " Respiratory exercises in Treatment of Disease, notably of the Heart, Lungs, Nervous and Digestive Systems," by Dr. Harry Campbell. London, 1899.

hyper-resonance *on* both *sides of the chest. The disease is* EMPHYSEMA.

§ 120. **Emphysema** is a chronic non-febrile disease of the lungs in which the air vesicles become hyper-distended, the walls separating each vesicle become atrophied, inelastic, and ruptured, and as a result the aërating surface is greatly diminished, and the lungs deficient in their elastic recoil.

Symptoms.—(1) The onset of the disease is imperceptible, and generally supervenes gradually after repeated attacks of bronchitis, the patient becoming more and more breathless after each attack. (2) This breathlessness is practically the only symptom, and it differs from all other kinds of breathlessness in this, that the chest remains *permanently in the inspiratory position*—in other words, owing to the inelastic state of the lungs and the shape of the chest, the patient finds it more difficult to expire than to inspire. A certain degree of cyanosis is generally present. (3) Symptoms of bronchitis are *nearly always present.*

The *Physical Signs*, expressed *briefly*, are a barrel-shaped chest, hyper-resonance, and prolonged expiration. The shape of the chest is special to emphysema (see Fig. 33). The chest assumes permanently the shape of a healthy chest in a position of deep inspiration. The antero-posterior diameter is considerably increased (see § 86). The hyper-resonance is always bilateral, and it obscures the dulness of the neighbouring organs —namely, the heart, the liver, and the spleen. These organs are also displaced downwards. The apex-beat may not be palpable, but epigastric pulsation is usually felt. On auscultation, the respiratory murmur is modified ; the inspiratory sound, which is full, is followed by a pause, and then by a prolonged expiratory sound. There are no adventitious sounds proper to emphysema, but, as just mentioned, bronchitis (*q.v.*) nearly always accompanies it. The heart sounds, especially at the base, may not be heard, or only with difficulty. Well-established emphysema interferes considerably with the pulmonary circulation, on account of the ruptured alveoli, and consequently the right side of the heart in course of time becomes dilated.

Variety.—In old people there is sometimes hyper-resonance with weak breath sounds, but no enlarged barrel chest ; this is called *Atrophic* Emphysema, and is due to the giving way of degenerate air vesicles.

The *Diagnosis* is extremely easy, because the bilateral hyper-resonance, the prolonged expiration, and the barrel-shaped chest are quite characteristic (*vide* Table of Diagnosis).

Etiology.—(i.) Emphysema occurs usually in elderly subjects. Both sexes are affected, but it is much commoner in males owing to the prevalence of bronchitis and asthma in them. (ii.) Heredity is said to play no part in the disease, but undoubtedly a hereditary tendency can frequently be traced. (iii.) Certain occupations render people prone to emphysema—*i.e.*, those which throw strain upon the lungs, as in the case of glass-blowers, wind-instrument blowers, etc. (iv.) The disease is frequently associated with senile degeneration, chronic Bright's disease,

TABLE IX.—CAUSES OF HYPER-RESONANCE.

Cause.	Hyper-resonance.	Auscultation.	Other Diagnostic Features.
I. **Emphysema.**	Bilateral and universal.	R.M. distinct and expn. much prolonged ; signs of bronchitis, if present.	Barrel-shaped chest, cardiac dulness obscured, and organs displaced.
II. **Pneumothorax**, mostly Hydropneumothorax. An acute condition.	Hyper-resonance always unilateral, though it may extend beyond middle line.	Absence of R.M. and V.F. over affected area ; sometimes amphoric breathing. Bell sound.	Organs displaced; history of emphysema or tuberculous cavity.
III. **Skodaic Resonance** —*i.e.*, the highpitched note above a large *pleuritic effusion*, when the lung is otherwise healthy.	Unilateral ; level may shift with position of patient.	Loud R.M. ; V.F. felt over affected area.	History of pleurisy ; signs of fluid lower part of chest
IV. A very **large cavity**, or extensive bronchiectasis (rare).	Unilateral, and of limited extent (may be cracked-pot sound).	Amphoric breathing, whispering pectorilloquy.	Expectoration of pus and long history of phthisis or bronchitis.
V. A **Tumour** (or pneumonic consolidation) between the chest wall and a large bronchus (rare).	Unilateral, and of limited extent ; dulness elsewhere.	Tubular breathing and bronchophony.	Symptoms of intrathoracic tumour.
VI. **Infiltration of solid** and even fluid material through the lung, *e.g.*, early stage of pneumonia, military tubercle, etc. (rare).	Hyper-resonance not marked (may be bilateral).	Signs of consolidation in some parts.	Hyper-resonance generally transient.

and cardio-vascular changes. (v.) Bronchitis is the most frequent of the exciting causes, owing to the prolonged coughing and straining to get up phlegm, and owing also to the blocking of certain tubes with thickened secretion, which prevents the access of air to some alveoli, and unduly distends others. (vi.) Asthma is also a potent exciting cause, owing to the constant strain on the elastic tissue of the lungs.

Prognosis.—Patients may live with emphysema to a good old age, and provided it is only moderate in degree it does not necessarily shorten life, though it predisposes to, and adds to, the seriousness of other pulmonary disorders. The gravity of any particular case is best measured by the extent of cardiac involvement (*q.v.*).

Treatment.—The indications are : (i.) To relieve the accompanying bronchitis (see § 108) ; (ii.) to improve the cardiac condition ; and (iii.) to restore as far as may be the elasticity of the lungs. The diet is of considerable importance in advanced emphysema, for any distension of the stomach greatly adds to the respiratory distress. It is a good rule never to let patients take a solid meal later than two o'clock in the day ; otherwise their nights become considerably disturbed by the breathlessness. Cardiac tonics, especially strychnine, and, in my experience,

tinctura cacti grandiflori, are useful.   Quinine and cod-liver oil often do good, although I cannot explain how the latter acts.

To restore the elasticity of the thorax is important, but difficult to accomplish ; of late years a special form of respiratory exercise has been put forward as fulfilling this condition, and to relieve the difficulty of expiration.   With this end in view, Gerrhardt has recommended the employment of mechanical expiration by compression of the thorax methodically, every day for five or ten minutes, by another person who places his two hands flat upon either side of the patient's chest.   A similar result has been attained by Rossbach's Athemstühl (breathing-chair).   A better method, when available, is the periodic use of a compressed air chamber.   In view of the fact that in many cases the maintenance of the inspiratory position is due to calcification of the costal cartilages, division of the latter has been attempted success- fully by some surgeons.

GROUP D.—There are three chronic pulmonary conditions in which the percussion note varies considerably in different cases, but the **offensive character of the sputum** reveals their presence—viz. : I. BRONCHIECTASIS ; II. GANGRENE ; and III. ABSCESS OF THE LUNG.   In Abscess the sputum is not so invariably offensive.   IV. ACTINOMYCOSIS and other diseases due to fungi affecting the lungs can usually be diagnosed only by examination of the sputum.

§ 121. I. **Bronchiectasis.**—Bronchiectasis is a cylindrical or saccular dilatation of the bronchial tubes.   The condition is met with most frequently as a complication of chronic bronchitis or chronic pneumonia.

Symptoms.—The patient complains chiefly of persistent cough.   At intervals of several days violent increase of coughing occurs ; it is started, perhaps, by some change of posture, and is followed by the expectoration of a large quantity of extremely fœtid sputum.   In the intervals the sputum is scanty, but the breath is offensive.   The fœtid sputum contains pellets or " Dittrich's plugs," and sets characteristically in three layers (see § 91).

The *Physical Signs*, if present, are mostly those of a cavity, attended by general signs of chronic bronchitis in both lungs ; occasionally only one is affected.   The patient is often cyanosed, and has clubbed fingers.

Diagnosis.—The extremely fœtid odour of the sputum—occurring as it does *at intervals* of perhaps several days or weeks, during which the sputum is not fœtid— distinguishes bronchiectasis from all other diseases.   In *gangrene* of the lung the sputum may be fœtid, although in a less degree, but it lacks the intermittent char- acter.   The position of the bronchiectasis is generally marked by dulness in the lower lobe of one lung which may be made to disappear by making the patient lie face downwards for some time, with his head low, till he coughs up a large amount of sputum.   The causes of the two affections also aid the diagnosis.   Abscess of the lung is attended by a very profuse purulent expectoration, but it is not so fœtid.

Etiology.—(i.) In patients past middle life by far the most usual cause is prolonged chronic bronchitis, and in children whooping-cough.   The dilated bronchial tube results from the continual strain of coughing on the weakened walls.   (ii.) Various forms of chronic pneumonia and chronic phthisis are believed to be attended by bronchiectasis, but in such cases it is probably a cavity in the lung tissue, and not true bronchiectasis, that we meet with.   (iii.) A foreign body plugging a bronchus is an occasional cause.   (iv.) In very rare cases it is a congenital defect.   (v.) After infectious diseases the condition of bronchiectasis may supervene.   (vi.) Tumour and syphilis.

Prognosis.—The condition is a very serious one, and for the most part incurable.   The patient may live from one to ten years.   The prognosis is much worse in bilateral cases, and in cases associated with extensive disease of the lungs or pleura.

The *Complications* which may occur are fatal hæmorrhage, gangrene of the lung, lobular pneumonia, and pyæmia.

Treatment.—The indications are to relieve the disgusting fœtor and to cure the primary disease. The first is accomplished by liberal antiseptic inhalations of turpentine, coal-tar, or creosote. The most valuable form of treatment is the creosote vapour bath. Patients are placed in an air-tight room, in which 30 drops of creosote are volatilised, the daily time of exposure being gradually increased from fifteen to forty-five minutes. The eyes and nose must be protected from the vapour. Terebene and creosote may be given in capsules (4 minims (0·2)) three times a day. Some inject menthol or guaiacol into the trachea in the proportion of 5 (0·3) and 2 (0·12) grains in 1 drachm (4) of olive oil twice a day. When the cavity is low down and near the surface, surgical measures for its drainage have been adopted.

§ 122. II. Gangrene of the Lung.—Owing to the extreme vascularity of the pulmonary tissues, gangrene of the lung is a rare condition, but it occasionally occurs over a limited area. It is usually a secondary condition, but it sometimes occurs in a lung previously healthy.

Symptoms.—(1) The onset may be acute, and marked by prostration and an irregular, intermittent pyrexia of a pyæmic type, with a very rapid pulse. (2) If, as is usual, the gangrenous part opens into the bronchi, a profuse fœtid expectoration soon follows. The sputum contains fragments of lung tissue, and generally blood also. The breath is extremely fœtid. (3) Pain in the side is usual, though it depends upon the involvement of the pleura. There is persistent cough, which aggravates the pain. (4) The *Physical Signs* are those of consolidation, sometimes those of a cavity.

Diagnosis.—The only condition which is liable to be mistaken for it, by reason of its fœtid expectoration, is bronchiectasis, which is distinguished by having (i.) " Dittrich's plugs " in the sputum, and (ii.) a gradual onset and longer course.

Etiology.—It is predisposed to by intemperance, old age, diabetes, and marasmus. Exciting causes are : (i.) Particles of food entering the lung, as in the insane, or patients with laryngeal paralysis, or persons in a drunken coma. In children a foreign body swallowed may produce it, though rarely. (ii.) Septic matter passing from the throat or mouth. (iii.) Severe asthenic types of pneumonia are occasionally so complicated. (iv.) It may complicate bronchiectasis. (v.) Septic emboli. (vi.) Aneurysm pressing on the root of the lung. (vii.) The extension of an abscess near the lung.

Prognosis.—The disease is almost invariably fatal, either immediately from collapse, sometimes from fatal hæmorrhage, or, later, from prostration. A few cases have recovered where the patch was of small extent. In cases which have been recorded as lasting for months or years it is very doubtful whether the lesion was true gangrene ; bronchiectasis is more probable. Occasionally the condition leads to pyopneumothorax.

Treatment.—Keep up the strength of the patient by means of abundant nutritive stimulants, iron and quinine. For the rest, the treatment is the same as in bronchiectasis. Surgery has succeeded in some cases.

§ 123. III. Abscess of the Lung is a serious and, happily, rare condition, but as it is nearly always secondary to some grave or fatal disorder, it does not add very materially to the gravity of the situation.

It is usually manifested by the expectoration of a large quantity of purulent pus which may or may not be fœtid, and is never so fœtid as in bronchiectasis or gangrene. The constitutional disturbance to which it gives rise is usually masked by that of the primary malady. The *Physical Signs* are those of localised consolidation, but these also are generally masked by those of the primary lesion. When the abscess bursts, the signs are those of a cavity.

It may occur in the course of (i.) advanced pulmonary tuberculosis ; (ii.) pneumonia ; (iii.) pyæmia ; (iv.) cancer, suppurating hydatid, or other tumours of the

lung, such as a gumma breaking down—a somewhat rare condition ; (v.) it occasionally follows the introduction of septic foreign bodies or wounds in the throat.

Prognosis.—The prognosis is very grave, but depends upon the cause. Occurring in the course of pyæmia, it indicates the progress towards a fatal termination. It is less grave in pneumonia occurring in otherwise healthy persons.

Treatment.—Medical treatment is not of much use. Surgical interference is not good in malignant and pyæmic conditions, but in other conditions, if fairly superficial, the abscess may be drained. Much progress has been made in surgical procedures for lung troubles during recent years. Artificial pneumothorax (p. 185) may be required.

§ **124. IV. Actinomycosis** may affect the pleura or the lung, imitating the signs of empyema, pneumonia (§ 100), phthisis, or bronchiectasis. In the absence of cutaneous or other lesions it is rarely diagnosed except by an examination of the sputum, when the little yellow pellets containing the ray fungus are visible. The blood serum gives a specific agglutinin reaction. The disease is usually fatal.

Aspergillosis. The fungus aspergillus fumigatus may cause signs resembling tuberculosis. The disease affects pigeon-feeders, who chew the seeds containing the fungus. It may undergo spontaneous resolution.

Blastomycosis and **Sporotrichosis** may affect the lungs. Cutaneous and other lesions are usually present in addition. **Ascaris** infection may be hard to diagnose in early stages.

CHAPTER VII

THE UPPER RESPIRATORY PASSAGES AND THE THYROID GLAND

The throat may be the seat of the same morbid processes as affect other mucous structures, such as catarrh, ulceration, or new growths. It is, moreover, in this position that several very important general or constitutional maladies, such as diphtheria, scarlatina, and syphilis, have important local manifestations. These facts have long been known, but it has come to be recognised only quite recently that the throat, and especially the tonsils—organs whose functions are still imperfectly known—may constitute the portal of entry of certain microbic conditions. It has also been suggested that the virus of influenza, rheumatism, malignant endocarditis, and other septic conditions, may thus gain admission into the general systemic circulation.

This chapter will deal with the symptoms referable to the **pharynx** (§ 125), the **larynx** (§ 138), the **nasal cavities** (§ 152), and the **thyroid gland** (§ 158).

THE THROAT

§ **125. Symptomatology.**—" The throat " may be said to consist of the fauces, tonsils, palate, pharynx, and larynx, and we are here concerned with the investigation of these structures. The symptoms indicating disease of these parts are principally two—namely, SORE THROAT and HOARSENESS. The examination of the mouth and tongue is described under Disorders of Digestive Tract (Chapter VIII).

(a) SORE THROAT is indicative mainly of disease of the *pharynx*, tonsils, and structures around. If the patient complains of " sore throat," turn to § 127.

(b) HOARSENESS AND OTHER ALTERATIONS OF THE VOICE are indicative of some affection of the *larynx* (§ 138). If NASAL INTONATION or NASAL DISCHARGE be present, turn to § 152.

There are also several minor symptoms which arise in conjunction with these, such as a dryness accompanied by tickling sensations, or an excessive secretion, which leads to " hawking " and " coughing." Thus it happens that we may be consulted for what the patient believes to be pulmonary disease, when in reality the lungs are perfectly healthy.

Dyspnœa and dysphagia may also be produced by local conditions of the throat and larynx. "Globus," a paroxysmal sensation as of a ball in, or constriction of, the throat is a symptom of hysteria.

§ 126. Clinical Investigation.—The anatomy and relations of the throat are indicated in Fig. 47 ; the various parts may be investigated by (a) direct, and (b) indirect (i.e., laryngoscopic) examination. .

(a) For the DIRECT EXAMINATION of the fauces and neighbouring structures all that is necessary is a good light and a spatula or spoon to
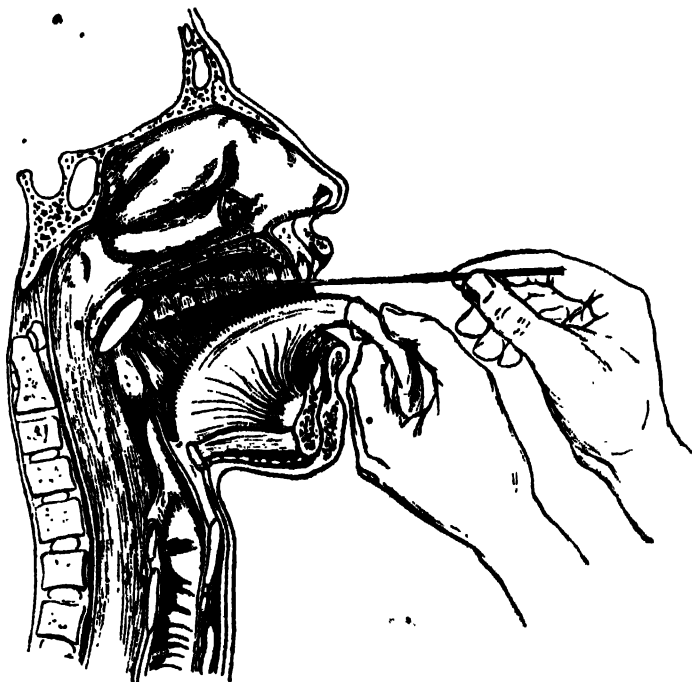
FIG. 47.--NASAL AND BUCCAL CAVITIES, showing the method of LARYNGOSCOPIC EXAMINATION— The three turbinate bones are seen, the anterior end of the inferior turbinate bone having been removed to show the inner opening of the lachrymal duct. The opening of the Eustachian tube is just behind the posterior end of the inferior turbinate bone. The exact position of laryngoscopic mirror in examination of larynx is shown—namely, over the root of the uvula.

depress the tongue. If direct light is not available—as for instance, when the patient is in bed—a laryngoscopic mirror can be used (vide infra), or a wax vesta, with or without a bright spoon behind it to act as a reflector. The patient should be instructed not to strain, and to " breathe quietly in and out." The posterior wall may be seen by directing the patient to say " Ha—ah," in which procedure the soft palate is raised. Note should be made of the colour of the mucous membrane, the presence of exudation or ulceration, of granulations or adenoid vegetations in the pharynx, of any mucous patches such as occur in syphilis, or any bulging

of the pharyngeal walls. The size and length of the uvula should always be observed, for a long uvula may be the sole cause of chronic cough and numerous otherwise unexplained symptoms. When a patient complains of cough coming on, or getting worse at night or when he lies down, elongated uvula should be suspected. It does not follow that such a uvula may appear too long at the time of inspection. Temporary congestion from various causes, e.g., much talking, produces undue elongation and nocturnal cough. Painting with tannin may reduce it, but the proper treatment is amputation, and it is wonderful what immediate relief is obtained.

(b) The INDIRECT or LARYNGOSCOPIC EXAMINATION of the 'throat is described in § 138.

§ 127. Classification, Diagnosis, Prognosis, and Treatment.—Sore Throat is a symptom common to nearly all diseases of the throat. Mentioned in order of frequency, the diseases which give rise to sore throat are as follows (*laryngeal affections being excluded for the present ;* see § 138) :

Commoner Causes.	*Rarer Causes.*
I. Catarrhal pharyngitis, including two acute and three chronic varieties.	VI. Retro-pharyngeal abscess or tumour.
II. Tonsillitis.	VII. Phlegmonous sore throat and acute œdema.
III. Scarlet fever.	
IV. Diphtheria.	VIII. Cancer, and other new growths.
V. Syphilis.	IX. Tuberculosis.
	X. Other acute specific fevers.

§ 128. 1. Acute Catarrhal Pharyngitis is an inflammation of the mucous membrane of pharynx and soft palate, and to a certain extent of the tonsils also. It may be so mild as to cause only slight discomfort in swallowing, dryness of the throat, tickling and hawking, and in such mild cases there is only a moderate congestion of the parts. But in severer cases there are constitutional symptoms of some severity, and locally there may be œdema and ulceration. The temperature in such cases varies from 100° to 104° F. The disease rarely lasts more than a few days, ending generally in resolution, although sometimes it passes into a chronic condition.   •

Hospital Sore Throat is a severe variety of the preceding, attended by considerable ulceration upon the fauces, tonsils, and even pharynx. There is the greatest difficulty in swallowing, speaking, and sometimes in breathing. The submaxillary and cervical glands are enlarged, and there is often considerable pyrexia and constitutional disturbance, the prostration being out of all proportion to the local inflammation.

(a) **Chronic Catarrhal Pharyngitis** presents the same symptoms as the acute variety, in a milder degree, and extending over a longer period of time. It is often known as Relaxed or Relapsing Sore Throat, on account of the chronic congestion of the parts and the consequent predisposition

to the repeated occurrence of subacute attacks.   It forms one variety of clergyman's or school-teacher's sore throat.

(b) **Granular (Follicular) Pharyngitis** is a *chronic* condition, the local symptoms of which resemble the foregoing, with the addition of visible granulations on the pharyngeal walls due to the enlargement of the follicles ; [1] hence it is sometimes called follicular pharyngitis.   This is a common condition, and a person who is the subject of it, although apparently in good health, is liable to suffer from repeated attacks of sore throat whenever the weather is damp or his health a little below par.   There is excessive mucous secretion, which collects in the throat, especially in the morning, and leads to chronic cough and hawking.   When the disease has lasted some time, the throat becomes dry from atrophy of the follicles (Pharyngitis sicca).

(c) **Granular (Adenoid) Pharyngitis** is another form of *chronic* pharyngitis,·due to the presence of adenoid hyperplasia and vegetations in the pharynx and naso-pharynx.   They are sometimes confined to the naso-pharynx, and by an ordinary inspection of the fauces little mischief, excepting congestion, can be discovered.   The lymphoid granulations may involve a large part of the naso-pharynx, occurring as a large grooved cushion or pedunculated growth, which, on examination, can be seen and felt behind the soft palate.   This condition is said always to start in childhood.   The child *breathes with the mouth open*, and thus acquires a characteristic vacancy of expression.   The intellect is.often below the average.   The voice has a dull or nasal twang, and there are snoring and disturbed sleep.   The nares are narrowed, and the palate may be high from the negative pressure in the nose, the diminished air tension in the nose not counterbalancing the normal air tension on the buccal aspect of the hard palate.   Pigeon-breast may follow.   The condition is a pregnant cause of middle-ear catarrh and subsequent deafness.   Adenoids in the naso-pharynx are usually accompanied by chronic enlargement of the tonsils.   The disease often runs in .families.

The *Causes of pharyngeal catarrh* vary somewhat in the different forms, although the several causes are largely interchangeable.   (1) There is no doubt that in certain persons exposure to cold and damp is immediately followed by an attack of pharyngitis, but this probably acts only as an exciting cause.   (2) Unhygienic surroundings, such as bad drains, the atmosphere of a hospital, and the like, may certainly give rise to a very severe ulcerating pharyngitis (*e.g.*, hospital sore throat) ; and here, again, the cause is of microbic origin.   The same condition may arise in private houses in which the drainage is out of order.   (3) Bad health in the individual affords undoubtedly a predisposition to the disease, and especially to granular pharyngitis, so much so that the throat in some persons

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[1] The word "follicle" is applied, not only to the lacunar glands or crypts in the tonsil, but also to the localised collections of adenoid tissue found in the posterior wall of the pharynx.   These latter, when enlarged, form the "granulations" of the granular pharynx.

constitutes a veritable barometer of the state of their health. (4) The gouty and rheumatic diathesis offer a predisposition to pharyngitis. (5) Various local conditions, such as nasal obstruction or insufficiency, leading to mouth-breathing ; and thus chronic rhinitis and *adenoid vegetations* are potent causes of recurring " sore throats." Wrong methods of production of the voice (clergyman's and school-teacher's sore throat), excessive smoking, the constant use of alcohol, spiced or hot foods, or working in a dust-laden atmosphere, often play an important part. The bristle of a tooth-brush or a fish-bone impacted in the pharynx is a not infrequent though unsuspected cause. (6) I have often met with chronic pharyngitis in people who live too well. The excessive secretion and the perpetual hawking have directed the attention of the patient and of his medical adviser to the throat, larynx, or lungs ; but the cure cannot be accomplished until dietetic and other measures are directed to the relief of the portal congestion. (7) Pharyngitis, especially in its chronic forms, is often associated with anæmia, and iron is one of the most valuable remedies we have. (8) The pharyngitis of influenza is slow to go, and is accompanied by a very irritating cough.

Prognosis.—Pharyngitis is one of the most frequent and troublesome of the minor ailments which we are called upon to treat. The milder varieties of the acute pharyngitis last only a few days, but the severer forms, such as hospital sore throat, may last many weeks, and be followed by considerable debility. All the chronic forms have a great *tendency to relapse.*

Treatment.—The indications are to relieve the local inflammation, to improve the general condition, and to prevent relapse. For the *acute forms,* most of the remedies mentioned under Tonsillitis are available. In all subacute and chronic forms, smoking, alcohol, and other causes of local irritation must be avoided. Excessive secretion may be removed by a gargle of bicarbonate of soda. For the " relaxed throat " a gargle consisting of a wine-glassful of water, to which a pinch of salt has been added, is useful ; so, also, are gargles of alum, potassium chlorate, and ammonium chloride (Formulæ 15 to 19). Probably carbolic acid, painted on as glycerine (B.P.), or employed as a spray, gargle, or lozenge, is the best application. A good spray is that of menthol (1 in 50 of paroleine). Later, astringent paints should be used—*e.g.,* nitrate of silver (4 per cent.) or equal parts of tincture of iodine and the glycerine of alum. Codeia in $\frac{1}{4}$ gr. (0·03) doses is the best remedy for the irritating cough.

The most efficient treatment for the granular forms of pharyngitis, where gargles are of little use, is painting with silver nitrate (8 or 16 per cent.), tannin (1 in 8), or with liquor ferri perchloridi, or iodine in glycerine. Trichloracetic acid is recommended highly. In cases of dry pharyngitis the ammonium chloride inhaler or lozenges are very useful. The actual cautery may be used to the individual granulations. All these measures, however, give only temporary relief to the adenoid variety, when scraping away the vegetations is necessary. For a permanent and radical cure

these must be thoroughly removed under general anæsthesia. Nasal obstruction, if present, must also be relieved. The general health in certain forms, especially the granular varieties, is often of more importance than the local condition, and many a relapsing and granular pharyngitis can be cured by Blaud's pills. Any rheumatic or gouty diathesis should receive attention, and dyspepsia or constipation, especially if associated with portal congestion, should be appropriately treated.

§ 129. II. **Tonsillitis**, or inflammation of the tonsil, is met with clinically in acute and chronic forms. *Peritonsillitis* is sometimes described as a variety ; it is an inflammation of the connective tissue in the vicinity of the tonsil; it accompanies catarrhal pharyngitis, and is sometimes due to decayed teeth.

The **three forms** of **Acute Tonsillitis** are as follows :

(a) **Acute Parenchymatous Tonsillitis** (Quinsy, Acute Suppurative Tonsillitis). The symptoms are pain, swelling and redness of the tonsils, coming on more or less suddenly with constitutional disturbance, the temperature varying from 101° to 104° F. Cases without pyrexia are occasionally seen. One tonsil is usually more affected than the other, and there is pain, stiffness, and tenderness behind the angle of the jaw. The disease usually subsides in the course of a week ; if it lasts longer than this suppuration has almost certainly occurred on one or other side. This is evidenced by the increased enlargement, by the swelling spreading along the soft palate, backwards, and downwards into the pharynx. The abscess usually bursts in the course of one or two weeks into the pharynx, but it occasionally points in other directions.

(b) **Acute Follicular Tonsillitis** is of a more superficial character. It is attended by the same symptoms as the foregoing, with the exception that abscess rarely occurs ; and the surfaces of the tonsils present numerous yellow points of thick purulent secretion, and perhaps ulceration. There are usually less fever and pain.

The *Diagnosis* of both these forms of tonsillitis from scarlet fever and diphtheria is sometimes a matter of considerable difficulty, but one of great importance. It is given in the form of a table (p. 209).

Etiology.—Even in healthy individuals many bacteria may be detected in the tonsils, e.g., staphylococcus, streptococcus, pneumococcus, influenza and micrococcus catarrhalis. In tonsillitis one or more of these may be the exciting cause. (1) Hereditary predisposition seems to play its part, for tonsillitis often occurs in subjects having a gouty or rheumatic tendency. Cold and damp weather are certainly conditions under which tonsillitis frequently arises. (2) Unhygienic conditions, and especially bad drainage, have been credited with causing the disease. (3) The tonsils become acutely inflamed in all cases of scarlet fever, in diphtheria, and in so large a proportion of cases of rheumatic fever that they are regarded as the portal of entrance of the virus of that disease. (4) Traumatism, such as drinking out of a boiling kettle. Fish-bones and bristles of a tooth-brush sometimes give rise to one-sided tonsillitis.

(c) A common form of acute tonsillitis is known as **Vincent's Angina**. It is often mistaken for diphtheria, with which it may be associated. It is characterised by one or more patches of exudation, often presenting a necrotic appearance, on the tonsils or adjacent anterior pillar, and sometimes encroaching on the palate. The pellicle is not easily detachable, and leaves a shallow ulcerated surface, the healing of which may be somewhat tedious. It is attended by some pyrexia and a variable amount of constitutional disturbance. Examination of a swab from the affected surface will reveal the presence of a large fusiform bacillus, which stains readily with the ordinary aniline dyes, but which will not grow on ordinary culture media, and a delicate mobile spirillum. Both these organisms, however, may be found occasionally in cases of ordinary ulcerative stomatitis, in carious teeth, and in some cases of septic scarlet fever. Perborate of soda, 1 in 4 of water, and iodine preparations form good antiseptic gargles.

Chronic Tonsillitis occurring in adults after repeated attacks of acute tonsillitis is a deep infection of the crypts, with or without any surface evidence. Sometimes crypt infection leads to a foul-smelling breath. In crypt infection of which the patient is unaware there may be set up a chronic toxæmia, leading to arthritis, neuritis, nephritis, and diminished vitality.

(a) **Chronic Enlargement of Tonsils** in children is generally associated with enlarged adenoids, and is regarded as a chronic hyperplasia. Except for the symptoms mainly due to adenoid enlargement (*vide* § 128), chokiness at night when lying down is characteristic. The supervention of an acute inflammation may give rise to considerable respiratory distress. This hypertrophic or obstructed type occurs as a rule in children of healthy appearance, rosy complexion, inclined to fatness, with a history of more or less freedom from the exanthemata. The occasional anæsthetic fatality described post mortem as Status Lymphaticus, occurs in this type of child. The chronic enlargement of tonsils and adenoids may be part of a general lymphatic hyperplasia, and the Status Lymphaticus, when it occurs, may be explained as an extreme type of this seemingly simple condition.

(b) **Chronic Tonsillitis** in children not associated with hypertrophy is of extreme importance. A local examination gives little information; the healthiest looking tonsil may be a veritable poison reservoir, acting as the primary focus in a general toxæmia. The condition is indicated by local and general symptoms. *Local:* running nose, muco-purulent rhinitis, postnasal discharge, fœtid breath, enlarged cervical glands, unilateral or bilateral otorrhœa, associated with a history of frequent colds and other infections, particularly the exanthemata. *General:* Pale, puny children suffering from nerve, respiratory or alimentary disorders, restlessness, night terrors, etc. In rheumatism and chorea the tonsils do not appear abnormal.

Course and Prognosis.—Acute tonsillitis is a frequent, and sometimes very troublesome, but never fatal, disease. Sometimes the patient continues at work, but at others he is totally incapacitated. Chronic tonsillitis is important, because it renders the patient liable to repeated attacks

of acute tonsillitis and coryza and is a common source of recurrent pharyngitis, leading to otitis media and deafness.. Enlarged tonsils met with in children occasionally disappear during adolescence; but in some way, only imperfectly explained, the mental and physical development of children who have chronic enlargement of the tonsils is sometimes impeded. It is, however, doubtful whether the development of the child is hindered unless there be concurrent adenoids, which interfere with the respiratory or oxidative processes of the body.

• *Treatment.*—The indications are (*a*) to reduce the local congestion ; (*b*) to reduce the pyrexia ; and (*c*) in chronic tonsillitis to prevent relapse.

(*a*) Powdered sodium bicarbonate applied directly to the tonsils has been credited with aborting the disease. A cocaine spray (4 per cent.) relieves the pain. Cold or hot compresses externally, steam inhalations, warm gargles of potassium chlorate, sodium bicarbonate, salol, and carbolic acid in glycerine (1 in 40) or formalin (2 per cent.) relieve the congestion (Formulæ 15 to 19). In subacute cases the tonsil may be painted with tannic acid, or tinct. ferri perchlor. in glycerine.

(*b*) To reduce the pyrexia a brisk saline purge should be given at the outset. Tincture of aconite (1 minim doses) may be given every half-hour during the first few hours ; then sodium salicylate, as in rheumatism, salol, or liquor ferri perchloridi. If quinsy does not clear up in a week, one may be almost sure an abscess has formed, and should be incised. This is best done with a curved bistoury, round which plaster is twisted to within half an inch of the point, which should be directed *inwards and backwards* to avoid the internal carotid. Make a small incision ; then insert a dressing forceps and stretch the opening. (*c*) In chronic tonsillitis the most useful remedies are iron, quinine, cod-liver oil, and other tonics. Salicylic acid, guaiacum, and colchicum are used in the relapsing form. Cauterising infected crypts may arrest the disease. Autogenous vaccines are advisable where relapses are frequent. The chronic enlargement may be diminished, by painting the throat with glycerine of tannic acid, " London Paste " or other astringents (*vide supra*) ; but in most cases the question of tonsillotomy or of enucleation of the tonsils arises sooner or later.

§ **130. III. In Scarlet Fever** the tonsil is generally the chief seat of inflammation in the throat. Both scarlet fever and acute tonsillitis start more or less suddenly, with constitutional symptoms, and thus the diagnosis is often one of considerable difficulty. There are four distinguishing features of scarlet fever—viz. : (1.) The diffuse *scarlet* colour of the soft palate and pharynx, with complete immunity of the larynx ; (ii.) sudden onset of the illness with high fever; (iii.) on the second day the rash ; and (iv.) about the third day the " strawberry " tongue (see Table X, p. 209, and § 380).

§ **131. IV. The sore throat of Diphtheria** may be recognised at once if there be an ashy-grey patch of exudation *upon the soft palate.* this is absent it is chiefly with follicular tonsillitis and Vincent's Angina

that difficulties arise. In diphtheria the large size and the colour of the patches (which are grey with surrounding red areolæ), the raised, sharply defined margin, the difficulty of removing them, and the raw bleeding surface left, enable us to come to a conclusion. The onset is more insidious, the pyrexia less marked, but the prostration is greater in diphtheria. A muco-purulent or hæmorrhagic discharge from the nose is very characteristic of diphtheria. Albuminuria is frequently observed with acute tonsillitis as well as with diphtheria. When other diagnostic features are absent, the presence of *one* large patch on a tonsil, instead of several small patches, is in favour of diphtheria. A swab will reveal the presence of the bacillus.

§ 132. V. **Syphilitic Sore Throat** is very characteristic. This and the other *secondary* manifestations of syphilis develop about three to six weeks after the appearance of the chancre. (1) The tonsils may be inflamed, but the inflammation is more generalised, and the mucous membrane presents greyish-white semi-translucent irregular patches (" snail-tracks "), on the fauces, tonsils, palate, and other parts of the buccal mucous membrane. Ulceration may also be present, especially on the tonsils, with red punched-out edges and yellow-grey exudation. Sometimes a raised white patch forms which teems with spirochætes. (2) Bilateral symmetry is a very characteristic feature of all these lesions.

Tertiary syphilitic ulcers may produce sore throat, their favourite position being the soft and hard palate, the tongue, and the fauces. They are usually preceded by gummatous swellings. (1) The ulcers are deep, with ragged floor, sharply cut edges, and covered with thick yellow-grey secretion. (2) They are progressive, and in course of time will destroy the hard palate or any other parts they invade. (3) They leave characteristic stellate cicatrices, which are indisputable evidence of the disorder.

The less frequent causes of Sore Throat are—RETRO-PHARYNGEAL ABSCESS, PHLEGMONOUS SORE THROAT, NEOPLASTIC ULCERATIONS, *and* ACUTE SPECIFIC FEVERS.

§ 133. VI. **Retro-pharyngeal Abscess,** or inflammation of the lymphoid and areolar tissue between the pharynx and the spine, may develop insidiously, or the onset may be comparatively sudden. It is known by (1) the rigidity of the head, with difficulty of swallowing and alteration of the voice ; (2) evidence of swelling in the posterior pharyngeal wall on inspection and palpation, by which means it is diagnosed from other causes of dyspnœa in children.

Etiology.—Those cases with an *acute* onset are generally either part of a septic inflammation after fevers, or occur in rachitic children under four. Retro-pharyngeal swelling coming on *slowly* is generally due to pus burrowing from some adjacent structure, especially from caries of the vertebræ.

Prognosis and Treatment.—The acute condition is always grave, and requires prompt surgical interference, generally free incision ; meanwhile, steam inhalations and warm fomentations relieve the symptoms.

TABLE X.

Tonsillitis.	*Scarlet Fever.*	*Diphtheria.*
	(a) LOCAL SIGNS.	
Swelling and redness chiefly confined to one or both tonsils. In the follicular form, tonsils covered with sticky mucus, with numerous small, separate yellow spots of secretion on one or both, which are easily removable. Nothing on soft palate.	Diffuse *bright* redness of throat and palate generally. The tonsils swollen, and may be covered with mucus and *sometimes* with multiple yellow points. Nothing on soft palate in ordinary cases.	Ashy-grey patch or patches on tonsils, uvula, *and soft palate* (latter situation is pathognomonic); patches *larger* than the follicular secretion in tonsillitis. Patches consist of membrane surrounded by red areolæ; difficult to remove, leaving raw surface. Klebs-Löffler bacillus found in membrane. Sometimes a muco-purulent, acrid *nasal discharge.* Comparative absence of pain.
	(b) GENERAL SYMPTOMS.	
(i.) O n s e t moderately sudden, with moderate fever. (ii.) Temperature may be very high, but local symptoms are usually more troublesome than general symptoms.	(i.) Onset very sudden, with high fever. (ii.) Temperature very high. Local symptoms a subordinate feature. (iii.) Rash on second day. (iv.) Strawberry tongue about third day.	(i.) O n s e t insidious. Early and marked enlargement of cervical glands. (ii.) Temperature not so high at first, and may remain low during whole course. (iii.) Paralytic sequelæ sometimes.

§ **134. VII.** PHLEGMONOUS SORE THROAT—*i.e.*, Acute Septic Inflammation of the Pharynx and Larynx—or ANGINA LUDOVICI (when the inflammation is chiefly external, in the neck).[1]—This very severe disease may start *inside* the throat, with symptoms of sudden pain, accompanied by considerable swelling, leading to severe dyspnœa, stridor, aphonia, and complete dysphagia in a few hours. There is much œdema around the fauces, followed by a brawny infiltration of the skin of the neck, spreading from under the jaw to the tongue and larynx. Sometimes the infiltration starts *externally*, and rapidly invades the internal structures. There is great constitutional disturbance, and a temperature of 102° to 105° F., but unless pus forms, rigors and delirium are generally absent. Pus formation is further indicated by widely and irregularly intermittent pyrexia. Mild cases begin with a stiffness and pain in the tissues around the jaw, and if recovery is to take place, the symptoms go no farther. But in many cases, and especially in alcoholic and debilitated subjects,

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[1] The disease has been variously described by the following names : Acute Inflammatory Œdema, Erysipelas of the Throat, Phlegmonous Cellulitis, Acute Infectious Phlegmon (a term applied by Senator when the inflammation was confined to the wall of the pharynx).

the disease rapidly progresses, and death takes place in twelve to forty-eight hours from heart failure, coma, or asphyxia from œdema of the larynx. Suppurative forms are very fatal. Among the recognised complications are pneumonia, pericarditis, pleurisy, and meningitis. There is a more chronic form in which induration is in excess of pus formation ; this may continue indefinitely until the pus is found and drained.

Etiology.—The condition, happily, is rare, and the causes consequently obscure. (1) It sometimes arises in association with scarlet fever, erysipelas, and small-pox (in former times being a common cause of death in this disease), or other acute specific fevers. (2) Dental suppuration or an alveolar abscess often forms the source from which rapid infiltration starts. (3) It may arise in people apparently in good health, and has then been attributed to the entrance of a specific microbe by the tonsils, or through the socket of an extracted tooth.

Treatment.—The indications are to control the inflammation, and to keep up the strength of the heart. Quinine (4 or 5 grains) should be given every four hours. Iron and digitalis are recommended. Remove carious teeth or stumps. Free and early incisions should be made into the œdematous tissues, and the practitioner should be at hand to perform tracheotomy if the dyspnœa be increasing. Stimulants must be liberally administered.

ACUTE ŒDEMA of the throat may be part of the above disease when the œdema is secondary to septic infection ; or it may be part of a general dropsy or angio-neurotic œdema. It is dangerous, as it may spread to the larynx and cause death by suffocation (§ 121a).

§ 135. VIII. CARCINOMA frequently, and SARCOMA occasionally, affect the pharynx, either primarily or secondarily. Their diagnostic features are the same as those mentioned under The Tongue (§ 170).

§ 136. IX. TUBERCULOUS ULCERS of the pharynx are rare as primary lesions. (1) They resemble syphilitic ulcers, but there is pallor of the mucous membrane, and a characteristic " worm-eaten " appearance of the pharyngeal wall. (2) *Their course is not nearly so rapidly progressive.* (3) It may be possible to obtain the tubercle bacillus from the scrapings ; and (4) there are usually other manifestations of tubercle, especially in the lungs. For treatment, see Tuberculosis of the Larynx (§ 146).

§ 137. X. ACUTE SPECIFIC FEVERS other than those mentioned above, such as typhoid, give rise to inflammation, and ulceration of the throat. In variola, for example, the pustules often form upon the palate, fauces, and buccal mucous membrane, leaving superficial circular ulcers. An examination of the throat is often useful as an aid to the diagnosis between measles, scarlet fever, and small-pox. The first named always affects the *larynx*, rarely the pharynx ; scarlet fever always affects the *pharynx*, and very rarely the larynx ; whereas small-pox affects them *both about equally.* Patches of *Lichen planus* may be found on the palate even before the disease occurs on the skin, and the eruption of varicella may be found in that situation. Other patches may be due to *thrush* or *herpes.*

The Larynx

§ 138. **Symptoms and Clinical Investigation.**—It will be remembered that the two cardinal SYMPTOMS of diseases of the throat (used in its widest sense) were (a) Sore Throat, and (b) Alterations of the Voice. Both of these may be present in disorders of the larynx, but it is the latter especially which indicates derangements of the organ of voice. Diseases of the larynx are also sometimes indicated by Cough, Hawking, Dysphagia, Dyspnœa, and actual Pain in the organ. But in some cases all of these may be absent ; there may, indeed, be pronounced disease of the

larynx (*e.g.*, paralysis or papilloma) without any *subjective* symptoms.[1]
• The CLINICAL INVESTIGATION of the larynx (laryngoscopy) is a proce-
dure of considerable technical nicety, and requires some practice. The
necessary appliances are a good steady light, a *reflecting mirror* mounted
on a band or a spectacle frame for the operator's forehead, and a small
circular *throat-mirror* mounted on a handle at an angle of 135°. The
light should be placed on a level with, and a little behind, the patient's
left ear. The operator takes his seat directly opposite ; and it is advisable
that his seat should be a little higher than that of the patient. Having
directed the patient to open his mouth and " breathe quickly in and out,"
the first step is to adjust the *reflecting mirror* in order to thoroughly illu-
minate the back of the pharynx. The focal length of the head-mirror
is generally 10 to 20 inches, and this should represent the distance of the
mirror from the patient's pharynx. Having warmed the throat-mirror
over the lamp to prevent the moisture of the breath from settling upon
it, the next step is to pull with the left hand the patient's tongue gently
out of the mouth with the aid of the corner of a towel or a piece of linen
rag. Take the corner of the towel in the right hand, lay it on the patient's
tongue, then grasp the tongue and towel firmly between the left thumb
and finger. Take care not to hurt the under surface of the tongue against
the teeth of the lower jaw. Then test the warmth of the throat-mirror
against your cheek or the back of your hand, and, having pushed the
patient's head a little backwards by pressing your right thumb against
the upper teeth, introduce the mirror with the right hand, *taking care
to avoid touching the top of the tongue* in so doing. Push the mirror obliquely
upwards against the soft palate just over its junction with the uvula
(Fig. 47, § 126). A good view of the vocal cords should be obtained
by slightly lowering or raising the handle. In children and persons with
very sensitive throats it is sometimes advisable to anæsthetise the pharynx
before laryngoscopy, either by a spray of, or painting with, a 4 or 5 per
cent. solution of cocaine, or by the administration of a few doses of
bromide during the preceding twenty-four hours.

In normal conditions the *epiglottis*, which is in reality anterior, appears
at the *upper part of the mirror*. The *vocal cords*, which are of a pearly
white colour, are close together at their upper or epiglottic ends ; and
at their lower (really posterior) ends are widely divergent during quiet
respiration. At their lower ends they appear to terminate in two promi-
nent knobs seen at the lower edge of the mirror, which mark the position
of the *arytenoid cartilages* (Figs. 48 and 48a). The *ary-epiglottic folds*
stretch on each side from the arytenoids to the sides of the epiglottis.
In these folds, just external to the arytenoid on each side, may be seen
a small prominence, the *cartilage of Wrisberg*. To the outer side of the

[1] Not long ago I met with the case of a well-known operatic singer who had a
small papilloma just beneath one vocal cord. Her voice was in perfect order, and
she could reach the highest notes with ease. The only defect was a hardly perceptible
weakness in the middle register.

cords lie the ventricular bands or false cords of mucous membrane. With a little practice, and under favourable circumstances, the bifurcation of the trachea may be seen.

DIRECT LARYNGO-SCOPY is now practised. By means of Killian and Brüning's bronchoscope or the modifications of it, the interior of the bronchi may be directly examined. When a foreign body has entered the air-passages, the patient should immediately be X-rayed, then examined by one who is expert in the use of this instrument.

Fig. 18 —Quiet Inspira-tion.   Fig. 48a —Forced Inspira-tion.

In LARYNGOSCOPY there are FOUR MATTERS to be investigated :

(a) The presence of *congestion* or *pallor* of the vocal cords and the parts around. Congestion of the vocal cords is an evidence of LARYNGITIS, sometimes of ulceration or new growth.

(b) The presence of any *ulceration.* Ulceration occurring in a person below middle age is very often due either to SYPHILIS or TUBERCLE ; in a person beyond middle life it is not infrequently MALIGNANT.

(c) The presence of a *nodule* or *new growth.* A nodule or new growth proves most frequently to be a PAPILLOMA.

(d) Whether there is any *paralysis* or *spasm* of the vocal cords, which is evidenced by the size, shape, and *mobility* of the aperture.

We shall deal with the disorders of the larynx in this order.

§ 139. Classification.- -As just mentioned, there may be no *subjective symptoms* even with pronounced disease of the larynx, and therefore it will be well to adopt as a basis of classification the *physical signs* discovered by laryngoscopy. However, when symptoms are present there is always some ALTERATION OF THE VOICE (except, perhaps, bilateral abductor paralysis, in which there may be dyspnoea and stridor without alteration of the voice). The principal diseases giving rise to such alterations (*i.e.,* the causes of alterations of the voice) may be grouped as follows :

   I. LARYNGITIS—
         (a) *Acute Laryngitis,* including also—
         Œdema Glottidis, and
         Foreign Bodies in the Larynx or Trachea.

         (b) *Chronic Laryngitis,* including also—
         Perichondritis, and
         Congenital Laryngeal Stridor.

   II. ULCERATIONS of the Larynx—
         (a) Tuberculous Ulceration,
         (b) Syphilitic Ulceration,
         (c) Malignant Ulceration.

III. Nodules and New Growth—

* (a) Benign,
  (b) Malignant.

IV. Paralysis of the Vocal Cords—

Bilateral Abductor Paralysis,
Unilateral Abductor Paralysis,
Total (Ab- and Adductor) Bilateral Paralysis,
Total (Ab- and Adductor) Unilateral Paralysis.

* V. Spasm of the Vocal Cords—

Laryngismus Stridulus (§ 151).

VI. Diseases of the Pharynx (§ 127); VII. Diseases of the Nose (§ 152); VIII. Some severe Pulmonary affections; and IX. Certain Neuroses also cause alterations in the voice.

I. *The patient complains of huskiness or loss of voice, a comparatively dry cough, soreness on swallowing, and there are local signs of congestion of the vocal cords. The disease is* LARYNGITIS, *of which two varieties are met with,* Acute *and* Chronic.

§ 140. **Acute Laryngitis** comes on somewhat rapidly, and usually runs its course in a week. As a rule it is not a serious affection, but in children it may be alarming. In children a slight laryngitis coming on suddenly is a frequent cause of what mothers describe as " croup." Owing to the dryness of the cords, the child wakes up suddenly at night with loud inspiratory stridor followed by an attack of coughing. This symptom is technically known as *laryngitis stridulosa*, and is not to be confused with laryngismus stridulus (see § 151). Simple laryngitis is differentiated from membranous croup (laryngeal diphtheria) by the perfect general health of the child in the former.

Etiology.—The chief cause of acute laryngitis is exposure to cold—especially when combined with overuse and wrong production of the voice (*e.g.*, actors, music-hall artistes, etc.). It is frequently a part of the " common cold." Diphtheria or measles may start in the larynx. Persons who suffer from chronic laryngitis (*q.v.*) or nasal obstruction are predisposed to attacks. A foreign body in the larynx or trachea is a cause of irritation which may produce symptoms resembling laryngitis.

Prognosis.—The affection is troublesome and apt to recur. When occurring during the course of the specific fevers, the prognosis is less favourable, because Œdema Glottidis may supervene.

Treatment.—All use of the voice must be forbidden. The patient must be kept in a warm, moist atmosphere, and should use warm inhalations (such as tr. benzoin co. ʒi. (4) to the pint (500) of boiling water, and see also Formula 110). Warm compresses or fomentations should be applied externally, and warm mucilaginous and alkaline drinks should be freely taken. The most efficacious medicine is one containing small doses of vinum antimonialis and potassium iodide. According to some, a strong solution of silver nitrate (1 in 3) applied locally at the outset may cut

short the disease. For laryngitis stridulosa, apply hot sponges to the throat, and give vin. ipecac. in teaspoonful doses, with warm water, every , ten minutes or so until emesis ensues. Adrenalin ♏ ii. (0·13) may relieve rapidly.

§ 141. Œdema Glottidis, or œdematous laryngitis, consists of an œdematous swelling affecting the epiglottis and submucous tissue of the larynx, but the vocal cords are not involved. The onset is usually sudden, and attended by considerable dyspnœa, dysphagia, and inspiratory stridor. The diagnosis is usually simple, on account of the swelling which can be seen and felt on palpation at the back of the tongue. · If this be absent, some difficulty may be experienced, but the sudden onset of laryngeal dyspnœa should bring the disease to our minds. It may arise either as a primary or as a secondary affection. As a primary disease it may come on as part of an acute septic inflammation of the throat, or it may be part of an œdematous angio-neurosis of urticarial origin (see Acute Œdema of the Tongue). It may occur as a *secondary* condition in association with (1) one of the various causes of acute or chronic laryngitis ; (2) a general anasarca ; (3) injury of the glottis by boiling or caustic liquids, etc. Its rapid onset is the chief source of danger, but if the patient does not shortly succumb to asphyxia, recovery generally takes place in a few days.

The *Treatment* consists in the administration of emetics, and ice internally and externally. In severe cases, if a 20 per cent. cocaine spray fail, scarification of the epiglottis must be resorted to ; and if this be unsuccessful, tracheotomy must be performed without delay.

§ 142. The Swallowing of a Foreign Body, and its passage into the larynx or trachea, has always to be borne in mind in children suffering apparently from acute laryngitis, for the history is often wanting. *Paroxysms of dyspnœa* or of *coughing* in a child without obvious cause should make us suspect it. Unless it has passed into the bronchus (usually the right), a foreign body may be seen by laryngoscopic examination. On the other hand, when a foreign body passes into the bronchus, it may cause so little cough or disturbance at the time that the patient may imagine he has swallowed it, or he may be unmindful of the incident. Some obscure cases of unilateral bronchiectasis are probably due to such causes. When the presence of a foreign body is suspected, a skiagram should be taken, and with the aid of direct bronchoscopy the object may be removed even from the bronchus.

§ 143. Chronic Laryngitis is a troublesome affection on account of the perpetual hoarseness and liability to acute laryngitis. Its causes are (1) repeated acute attacks ; (2) excessive speaking, singing, teaching, and overuse of the voice (actors, clergymen, school-teachers, etc.) ; it also affects masons, fustian-cutters, and others exposed to dusty atmospheres ; (3) nasal obstruction and mouth-breathing ; (4) tubercle, syphilis, and new growths, evidences of which should always be sought for, in cases of intractable laryngitis. These usually go on to ulceration, under which they will be described. (5) Spread of inflammation from adjacent parts. Many cases of chronic laryngitis depend upon a granular condition of the pharynx. (6) Rheumatic and gouty diatheses predispose.

Treatment.—The indications are to avoid the cause and to relieve the local congestion. The removal of the cause is most important, and often most difficult to accomplish, for a large number of the patients are singers, teachers, and others whose living depends upon the daily excessive use of the voice. The avoidance of tobacco and alcohol will aid, and residence in a dry climate will often accomplish a speedy cure. Much may be done

to prevent or relieve the condition by proper voice-production and respiration. This affection is extremely common among our board-school teachers, owing chiefly to faulty voice-production, and they ought to be specially trained to obviate this defect. Locally, painting with strong astringent remedies, such as zinc chloride (1 in 16) or silver nitrate (1 in 24 or 1 in 16), are useful. These strong applications should not be made more than twice a week ; weaker solutions can be applied more frequently. The patient himself may use sprays of alum (1 per cent.) or zinc sulphate (½ per cent.) for five minutes twice daily, or inhalations of turpentine, creosote, iodine, menthol, etc., for fifteen minutes three times a day.

§ 144. **Perichondritis** is an inflammation of the perichondrium of the laryngeal cartilages. Opinions differ as to its frequency. If considerable, it may lead to necrosis of the cartilages and abscess of the larynx. The differential features, besides loss of voice or hoarseness, are dull aching pain and acute tenderness. This may be accompanied by swelling in the neck. As regards its *Etiology*, apart from traumatism, it is rarely a primary malady. It more often occurs secondary to syphilitic or tuberculous laryngitis. Syphilis is its commonest cause. It also follows enteric fever.

Prognosis and Treatment.—It is a serious affection, for even in the mild forms the voice is rarely restored. Great stenosis of the larynx may result. If there be much swelling, the dyspnœa is very marked, and the patient may die from pneumonia or gangrene of the lungs, or, in the suppurating forms, from pyæmia. Abscess and fistula may follow.

§ 145. **Congenital Laryngeal Stridor** is a term applied to more or less continuous inspiratory dyspnœa, accompanied by a croaking sound, occurring in infants. It may be constant up to the age of two, or occur only at intervals during that period of life. It is believed to be caused by a folding of the epiglottis, possibly due to some malformation. It is usually attended by a certain amount of laryngitis and hoarseness, and passes off without need for operative interference.

II. **Ulcerations** *of the larynx are met with chiefly in tubercle and syphilis, and in persons past middle life malignant disease may be a cause. The simple erosions present in catarrhal laryngitis hardly amount to ulceration. Ulceration is also found in the later stages of Lupus and Leprosy, usually when cutaneous lesions are present.*

§ 146. **Chronic Tuberculous Laryngitis** should always be suspected when delicate patients complain of constant hoarseness. This form of laryngitis is recognised by (1) the general pallor of the mucous membrane, accompanied by a thickening or swelling most marked over the arytenoids or the aryteno-epiglottic folds ; (2) the occurrence of irregular, slowly growing ulcers, usually bilateral ; and (3) the history or presence of pulmonary tuberculosis.

The *Prognosis* is always grave, and until recently recovery when the larynx was involved in tuberculosis was practically unknown. The course of the affection depends more upon the condition of the lungs (§ 110) than that of the larynx.

The *Treatment* at first is largely constitutional—*e.g.*, creosote in doses of 1 to 5 (0·06–0·3) minims is recommended. Locally, menthol, one part to five of olive oil, used as paint, or an insufflation of menthol (1 in 7) with equal parts of iodoform and boracic acid, is valuable. When ulceration has occurred, after being swabbed with cocaine and curetted, the parts should be thoroughly brushed with lactic acid, 10 to 60 per cent. This is a very favourite application. For the pain, which may be severe enough to cause dysphagia, morphia ¼ grain (0·016), with starch ½ grain (0·03), may be blown into the larynx ; or it may be sprayed with 10 per cent. cocaine. Dundas Grant injects alcohol into the superior laryngeal nerve with excellent results. Absolute rest from speech, a warm, dry climate, and sanatorium treatment, are indicated (§ 110).

§ 147. **Chronic Syphilitic Laryngitis.**—The laryngitis accompanying secondary

syphilis may resemble simple catarrh, with the addition of whitish patches (§ 132). But that which occurs in the later stages nearly always takes the form of ulceration. The intensity of hyperæmia, the irritability, and the profuseness of the purulent discharge are features of syphilitic ulceration. It is distinguished from a tuberculous ulceration by (1) the bright red coloration of the mucous membrane ; (2) the presence of a deep, *rapidly growing ulcer*, with bright yellow surface, regular edges, often underminded, sometimes unilateral. If the ulcers invade the upper surface of the epiglottis this is said to be pathognomonic of syphilis. (3) The presence of a syphilitic history.

Prognosis and Treatment.—This form of laryngitis is twice as rapid as, and far more destructive than, the preceding, and is liable to involve the cartilages (*vide* Perichondritis). Even when arrested considerable stenosis may result. The usual constitutional treatment must be carried out. Where salvarsan cannot be given, full doses (60 to 100 grains) of potassium iodide must be taken. Local applications of iodoform, or a spray of perchloride of mercury (1 in 1,000), are employed.

(c) **Malignant Disease** and (in other countries) **Leprosy** give rise to ulceration of the larynx (see below).

III. **Nodules and New Growths.**—*Flat localised thickenings of the mucous membrane are spoken of as warts, nodes, or nodules. When they are pedunculated they are spoken of as polypi. In either case they begin most frequently as a unilateral thickening on or near one of the* vocal cords. *In the early stage they are extremely difficult to distinguish from syphilis or tubercle, and sometimes this can be accomplished only by the history. With one important exception (singer's node) nodules are* UNILATERAL, *and this feature of asymmetry distinguishes them from the thickening which may result from chronic laryngitis. The practical point of prime importance is the distinction of benign from malignant growths, often a task of considerable difficulty.*

§ 148. **Benign New Growths** begin most frequently as **warts**, nodules, or thickenings, the surface of which is smooth although congested. They may give rise to no symptoms for a considerable time, unless they happen to be on the free edge of the cord. Perhaps the commonest of these growths is what is known as a **singer's node.** This lesion very often affects the under surface of the vocal cord, and hence may be overlooked for a long time. It is distinguished from other nodules by its frequent involvement of both sides symmetrically. A projection on one cord at the junction of the *anterior with the middle third* is probably a Singer's Wart ; one situated at the junction of the *posterior with the middle third* is probably pachydermia laryngis. In the latter case there is a nipple on one cord which fits into a crater on the other. **Pachydermia Laryngis** is a localised chronic laryngitis (§ 143), usually most marked over the vocal processes. *Benign* nodules, as a class, are differentiated from *malignant* by the absence of pain and the paucity of symptoms of any kind. A pedunculated benign growth (**polypus**) of the larynx has the same clinical features, but is accompanied by very characteristic attacks of paroxysmal dyspnœa. **Leprosy** may affect the larynx. Benign growths often cause but little inconvenience. They are generally removable, without ulterior damage, by snares or cutting forceps.

§ 149. **Malignant Growths** of the larynx occur chiefly in men. They may be divided into two groups, extrinsic and intrinsic. The *extrinsic* variety start as thickenings of the mucous membrane, which may resemble benign growths, or may be greyish-white, or have a rugged surface. It rapidly passes on to ulceration, with hæmorrhage and pain ; secondary enlargement of the glands follows. Death ensues unless the larynx is extirpated early. *Intrinsic* cancer, on the other hand, is of slow growth, and low malignancy. It usually starts in the vocal cord, and causes a persistent huskiness. Every case of persistent hoarseness occurring in men over middle age should be sent to a laryngologist for examination. The operation of laryngo-fissure affords 80 per

cent. of cures in these cases if seen early. - X-ray and radium therapy affords aid in certain cases.

IV. **Paralysis of the Vocal Cords** *can be detected only by carefully inspecting both the* POSITION *and the* MOBILITY *of the cords during* (i.) *rest,* (ii.) *phonation and* (iii.) *deep inspiration.*

§ 150. **Paralysis of the Vocal Cords.**—The chief actions of the larynx are (i.) Abduction (glottis-opening), which is performed by the posterior crico-arytenoids, and (ii.) Adduction (glottis-closing), which is performed by the lateral crico-arytenoids and the arytenoideus muscle. The cords are rendered tense by the crico-thyroids (external tensors), and are relaxed and shortened by the thyro-arytenoids (internal tensors—*i.e.*, the muscle which lies in the vocal cord). The larynx is supplied by two nerves, the superior laryngeal and the recurrent laryngeal branches of the vagus. The former supplies the crico-thyroid or tensor muscle and the mucous membrane of the larynx, while the recurrent laryngeal supplies all the other muscles. In progressive lesions of the recurrent nerve the abductors are paralysed first, and later on the adductors.

TABLE XI.

| Name of Muscle. | Nerve Supply. | Action.. | |
		Phonation.	Respiration.
Crico-thyroid or external tensor.	Superior laryngeal.	Tense and elongate the vocal cords.	—
Thyro-arytenoid proprius [1] *internal tensor in cord itself.*	Recurrent laryngeal.	Adjusts edges of the cords.	—
Posterior crico-arytenoid.	Recurrent laryngeal.	—	Abduct—*i.e.*, open glottis.
Lateral crico arytenoid.	Recurrent laryngeal.	—	Adduct—*i.e.*, close glottis.
Arytenoideus.	Superior laryngeal and recurrent laryngeal.	Close the glottis (posterior third chiefly).	—

The Signs of Laryngeal Paralysis.—It is very rarely that a single muscle is paralysed ; the paralysis nearly always affects a physiological group of muscle—*i.e.*, the glottis-openers (abductor paralysis) or glottis-closers (adductor paralysis) on one or both sides. Paralysis is often accompanied by more or less catarrh, which modifies the appearance somewhat, but the evidences of laryngeal paralysis depend upon the position and mobility of the cords during phonation and respiration. The symptoms are given in Table XII.

Normally, during rest the cords are midway between open and closed (Fig. 49) ; during phonation they are approximated so that practically no space is left between them (Fig. 51) ; during deep inspiration they are widely opened (Fig. 48a).

When the cords are normal during phonation, but do not move out on inspiration, there is bilateral paralysis of the glottis-openers—*bilateral abductor paralysis* (Fig. 52). If both cords move during phonation, but one of them fails to move out fully during inspiration, there is *unilateral abductor paralysis* (Fig. 53).

When the cords neither move to the middle line with attempted phonation, nor move as far outwards as normal during deep inspirations, but remain midway between

Lateral thyro-arytenoid is the lateral part of this muscle.

the two in the cadaveric position (Fig. 50), there is *total bilateral paralysis of* adductors and abductors (Fig. 54).

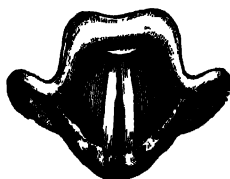If during phonation and inspiration one cord remains immobile, there is *total unilateral paralysis.*

FIG. 49.—MODERATE AD-
DUCTION.—The appear-
ance seen during REST.

FIG. 50.—CADAVERIC POSI-
TION of cords.

FIG. 51.—Typical position
during PHONATION of high
notes.

TABLE XII.—LARYNGEAL PARALYSES.

· (From Gowers, slightly modified.)

Lesion.	Symptoms.	Signs.
Bilateral abductor (opener) paralysis.	Voice little changed ; cough normal ; inspiration difficult and long, and attended with loud stridor.	Both cords near together ; not separated during inspiration, but even drawn nearer together.
Unilateral abductor (opener) paralysis.	Symptoms inconclusive ; little affection of voice or cough. Brassy cough sometimes.	One cord near the middle line not moving during inspiration, the other normal.
Total bilateral paralysis.	No voice ; no cough ; stridor only on deep inspiration.	Both cords moderately abducted and motionless (i.e., the cadaveric position).
Total unilateral paralysis.	Voice low-pitched and hoarse ; no cough ; stridor absent or slight whilst breathing.	One cord moderately abducted and motionless, the other moving freely, and even beyond the middle line in phonation.
Bilateral adductor (closer) paralysis.[1]	No voice ; normal cough ; no stridor or dyspnœa.	Cords normal in position, and moving normally during respiration, but not brought together on an attempt at phonation.

If there is aphonia, and on laryngoscopic examination the cords do not meet properly during attempted phonation, although they move outwards with inspiration, there is *bilateral adductor paralysis* (Figs. 55 and 56).[2]

The *Etiology* of laryngeal paralyses differs considerably in the various forms. They may arise from ORGANIC or FUNCTIONAL conditions, but each is so characteristic that it can be readily identified. Thus *hysterical paralysis* is always double, and very nearly always due to adductor paralysis. Abductor paralysis is generally—and if unilateral is always—organic in origin. If the left vocal cord cannot be abducted, it is almost certainly due to pressure on the left recurrent laryngeal, and this in nine cases out of ten is due to *aneurysm of the aorta.*

(a) BILATERAL ABDUCTOR PARALYSIS (Fig. 52) may be due to—

(i). The earlier stages of *pressure* upon both recurrent laryngeal nerves, as by mediastinal tumour, or pericardial effusion (§ 42).

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[1] *Unilateral* adductor paralysis is practically unknown, though tuberculous or rheumatic ankylosis of the crico-arytenoid joint may cause immobility of one cord.

[2] Further particulars of the actions of the various muscles may be found in a study of laryngeal paralyses since the introduction of the laryngoscope, by Sir Felix Semon (*Brain,* 1892, vol. xv., p. 471).

(ii.) *Peripheral* neuritis from toxins (such as diphtheria, alcoholism, influenza), certain drugs (*e.g.*, lead, arsenic), or simple catarrh.

(iii.) *Central Causes*, as in lesions affecting the medulla or base of the brain, bulbar paralysis, cerebral tumours or syphilis, hæmorrhage into the bulb, tabes dorsalis, disseminated sclerosis, meningeal conditions, etc.

(*b*) UNILATERAL ABDUCTOR PARALYSIS (Fig. 53) is due to the same causes acting on one side only. Thus, if on the *left side*, it is due in nine cases out of ten to aneu‧ rysm *of the aorta*, although no other signs of that condition may be present. Malignant tumour of the œsophagus may also affect the left recurrent laryngeal. Thickened right pleura may be the cause of a paralysed right recurrent laryngeal. Pressure upon the vagus in the neck, as by an enlarged thyroid, or cervical glands, may affect one or both sides.

(*c*) TOTAL (AB- and ADDUCTOR) BILATERAL PARALYSIS (Fig. 54) is practically always of organic origin, but it may (rarely) be due to catarrh or hysteria. It may arise from any of the causes mentioned under Bilateral Abductor Paralysis, but is most frequently of *central* origin. It occurs later in the disease than abductor paralysis, the abductor fibres in the nerve being the first to be affected.

(*d*) TOTAL (AB- and ADDUCTOR) UNILATERAL PARALYSIS is due to the same causes as mentioned under unilateral abductor paralysis—*i.e.*, usually pressure upon the recurrent laryngeal. This condition, however, occurs at a later stage in the case, unilateral abductor paralysis being a feature of the earlier stage. Total paralysis is sometimes called "recurrent paralysis," because it is due to paralysis of the recurrent laryngeal.

FIG. 52.—BILATERAL PARALYSIS OF THE GLOTTIS-OPENERS (BILATERAL ABDUCTOR PARALYSIS).—The patient is able to oppose the cords during phonation, but *the cords do not move outwards during deep inspiration* (as in Figs. 48 and 48*a*).

The same appearance as the above is sometimes produced by acute laryngeal catarrh, but the cords would be pink instead of white.

(*e*) BILATERAL ADDUCTOR PARALYSIS (Figs. 55 and 56) is always *functional* (viz., unconnected with *gross lesions*): (1) hysterical; (2) simple catarrh, or over-use of the voice; (3) general weakness, as in anæmia. But the first of these is by far the most common.

Prognosis.—Laryngeal paralysis is generally only a minor element in the case. When occurring alone, however, the prognosis in adductor paralysis is good, because it is always of functional origin. Paralysis arising from syphilis is remediable if treated early. In all forms, however, the prognosis depends upon whether the cause is removable or not.

Treatment.—Hysterical paralysis should be treated on lines laid down elsewhere. Strong faradisation or static electricity to the larynx is indicated, the patient being instructed to call out loudly. In organic paralyses the prognosis depends upon the cause. Syphilitic remedies should receive a fair trial. Strychnine and electricity are useful. In organic cases, if dyspnœa be severe, tracheotomy must be performed.

ILLUSTRATIONS OF LARYNGEAL PARALYSIS.—It should be remembered, in studying these illustrations, that to test the motor power of the vocal cords it is necessary to make the patient INSPIRE deeply to OPEN the cords, then to PHONATE, so as to CLOSE the cords, for a given position of the cords conveys no information unless it is first known which of these acts the patient is performing.

In laryngeal paralysis it is very important to decide whether a functional or organic cause is in operation, and the following hints should be remembered :

　　1. Glottis-closer (adductor) paralysis is generally functional; Glottis-Opener (abductor) paralysis generally Organic.
　　2. Bilateral paralysis is generally functional; One-sided paralysis is generally Organic.
　　3. Left Abductor (glottis-opener) paralysis suggests Aneurysm.

PATIENT'S RIGHT

PATIENT'S LEFT

FIG. 53.—LEFT ABDUCTOR, or glottis-opener, paralysis.—DURING INSPIRATION the *left cord* remains fixed, instead of moving outwards like the right cord does. This occurs in early paralysis of the recurrent laryngeal nerve of ORGANIC ORIGIN—*e.g.*, aneurysm.

FIG. 54.—TOTAL BILATERAL paralysis.—DURING INSPIRATION and DURING PHONATION *both cords* are immobile, and remain in what is practically the cadaveric position. Nearly always of ORGANIC origin, and frequently central.

FIG. 55.

FIG. 56.

FIGS. 55 and 56.—PARTIAL BILATERAL ADDUCTOR, or glottis-closer, paralysis.—It is the condition commonly met with in hysterical or FUNCTIONAL aphonia. DURING PHONATION the cords close anteriorly and posteriorly, but leave an elliptical space between them. The glottis is closed by two muscles—the crico-thyroid in front, and the arytenoideus behind. If the CRICO-THYROID is mainly affected, the condition depicted in Fig. 55 is seen, and it is met with in functional aphonia and exhaustion. The ARYTENOIDEUS closes the posterior angle, and when this is paralysed the posterior angle remains open (Fig. 56). Both of these forms are met with in acute and chronic laryngitis, and are generally independent of any actual nerve lesion, excepting perhaps peripheral neuritis and some rare cases due to a local lesion affecting the recurrent laryngeal nerve of both sides.

V. SPASM OF THE LARYNGEAL MUSCLES, *and consequent* INSPIRATORY DYSPNŒA, *is not a very common occurrence, except in the form of Laryngismus Stridulus, a disease almost confined to childhood. It may arise when a foreign body passes into the larynx, and may occasionally occur in adults who are the subjects of acute laryngitis. Inspiratory dyspnœa may also arise in Bilateral Abductor Paralysis.*

§ 151. **Laryngismus Stridulus or Nervous Croup** [1] is a form of paroxysmal inspiratory dyspnœa. It consists of a sudden spasmodic closure of the glottis, followed by a long noisy inspiration which produces a crowing sound, and is due to spasm of the adductors. It is a nervous affection, and appears to be due to some irritation of the vagus or of its recurrent laryngeal branch. The whole attack lasts from a few seconds to a minute or two. The child may become cyanosed or the spasms may spread to other muscles and give rise to general convulsions. Occasionally it terminates fatally. The attacks come on either during sleep, or in the waking state. They are very apt to recur, and the severity of the attacks may increase at each recurrence. On the other hand, if the attacks are slight, they may gradually disappear as the child grows older. In the intervals the child is free from cough or hoarseness, and the larynx appears healthy.

· [1] Synonyms : Spasmus glottidis, spasmodic croup, child-crowing, spasm of the larynx.

The *Etiology* is obscure. It is practically confined to children of from four months to two years old, and nine-tenths of these are rachitic—that is to say, children in whom infantile convulsions and tetany are also apt to arise. It is twice as common in boys. It is more frequent in the spring-time, and it is often hereditary. In older subjects laryngeal spasm and inspiratory dyspnœa occur sometimes in tabes dorsalis, when it forms the laryngeal crisis of that disease. Its rarer causes are epilepsy, hysteria, tetany, chorea, reflex irritation of the vagus or its recurrent laryngeal branch from mediastinal growths, a growth or foreign body in the larynx, or too long a uvula.

The *Diagnosis* is not difficult, though it is well to bear in mind the possibility of a foreign body in the throat, larynx, or trachea. There are, however, three pathological conditions to which the term " croup " is loosely applied and which are also characterised by a PAROXYSMAL INSPIRATORY DYSPNŒA.

1. *Laryngismus stridulus* is the non-inflammatory nervous affection described above. This is recognised by the absence of cough, hoarseness and other symptoms referable to the larynx in the intervals between the attacks. There is often a history of similar attacks.

2. *Catarrhal Laryngitis* (laryngitis stridulosa, false croup) is often associated with attacks of dyspnœa, coming on usually at night in children under ten who are suffering from cough and hoarseness during the day. It may last for an hour or so. This is due to the collection of thick secretion, or to the sticking together of the edges of the glottis from slight laryngeal catarrh (§ 140).

3. *Membranous Croup*, or laryngeal diphtheria.—This is true diphtheria, and is attended by the constitutional and other symptoms of that disease (Chapter XV). However, some (*e.g.*, Whitla and others) maintain that a non-diphtheritic membranous croup may occur. A severe injury (*e.g.*, drinking out of a boiling kettle) may certainly result in a membranous or·" diphtheritic " inflammation of the mucous membrane.

Treatment of Laryngismus Stridulus.—(*a*) *For the Attacks.*—In severe cases cold water may be dashed in the face, or the patient plunged into a hot bath, or alternately hot and cold, or cold water douches applied. Inhalation of chloroform or ether relieves it promptly. Artificial respiration is often of great service, and it may restore, even after apparent death. In the rare cases in which the spasm is prolonged and continuous, tracheotomy may be necessary. Mild cases require no treatment except rest and warmth.

(*b*) *For the Intervals.*—The patient should be kept very quiet, and irritation of the surface or the application of any stimuli conducive to an attack should be avoided. Reflex causes of irritation should be sought for in the gums (*e.g.*, teething), in alimentary canal (*e.g.*, worms or gastric disorder), in the lungs and elsewhere (*vide* causes). The general treatment of rickets should be adopted, and it is worth bearing in mind that children taken into the country very often cease to have these attacks. Sponging

with cold water twice or three times a day is of value ; and as to medicine, bromides and chloral in small doses allay the irritability of the nervous system, on which the condition mainly depends. Faradisation of the pneumogastric is sometimes useful.

VI. and VII. **Diseases of the Pharynx** (*ante*) **and of the Nose** (*post*) *are generally attended by a certain amount of hoarseness and alteration of the voice.* The latter give to the voice a peculiar nasal twang, which is very characteristic.

The Nasal Cavities

§ 152. **Symptoms and Physical Examination.**—Diseases of the nose will be considered under three cardinal SYMPTOMS : *Inodorous discharge* from the nose (Rhinorrhœa) ; *foul discharge* from the nose (Ozœna) ; *mouth-breathing* and snoring (Obstruction of one or both Nostrils). *Bleeding* from the nose also occurs in some nasal disorders, but it is *not* a cardinal symptom. It is perhaps more generally associated with some constitutional or general derangement. *Sneezing, tickling* in the nose and *sniffing* may also be present ; and the quality of the *voice* may be altered, particularly in nasal obstruction. The sense of *smell* is always disturbed to some extent in nasal disorders. In some instances, headache, vertigo, and other nervous derangements are met with in association with disorders of the nose, especially when the free transit of air through the nasal passages is interfered with, and the atmospheric pressure within the tympanum disturbed.[1] Various constitutional symptoms may result from septic conditions of the nose or the adjacent sinuses, and not infrequently a patient suffers from listlessness and general debility for a long time before our attention is directed to the true source of his troubles.

FIG. 57.—NASAL SPECULUM.

Clinical Investigation.—Rhinoscopy or examination of the nose may be effected through the anterior nares (anterior rhinoscopy), and the posterior nares (posterior rhinoscopy) ; and by digital examination posteriorly.

ANTERIOR RHINOSCOPY.—First examine the anterior nares for any obvious disorder, such as fissures, ulcers, scars from ulcers, any narrowing of the nares, or a deviation of the septum ; secondly, introduce a speculum (Fig. 57), using either a direct light or one reflected from a mirror on the forehead, as in laryngoscopy. In this way an examination of the inferior turbinate bone can be made, to see if it be hypertrophied. The inferior or middle meatus can be thus examined for polypi or alteration in the mucous membrane. If, as frequently happens, the anterior part of the inferior turbinate is hypertrophied, and hides the view, this may

[1] A notable instance in my own experience was that of a lady of thirty-five who suffered from the most troublesome tinnitus aurium and occasional giddiness, which was not relieved until the middle turbinate bone was removed. (See the Author's " Clinical Lectures on Neurasthenia," fourth edition.)

be reduced by swabbing out with a cotton-wool pledget soaked in a
10 per cent. solution of cocaine.

POSTERIOR RHINOSCOPY is effected by precisely the same procedure
as in laryngoscopy (§ 138), using the smallest of the mirrors, and turning
it upwards. It is convenient to have a special mirror for this purpose
mounted on a curved handle. The stem being hinged at its extremity,
so that it can be raised to any desired angle. It is important to avoid
touching either the dorsum of the tongue or the posterior wall of the
pharynx. The patient should be instructed to breathe gently all the
while through the nose. This depresses the soft palate and widens the
field of observation. By moving the mirror slightly in different directions
we are able to examine the posterior nares and turbinate bones, the inner
end of the Eustachian tube for any swelling, and Luschka's tonsil (*cf.*
Fig. 47). The pharyngeal or Luschka's tonsil is a mass of lymphoid tissue
on the pharyngeal roof and posterior wall above and between the Eusta-
chian tubes ; when in a condition of hyperplasia it forms the cushion-like
growth of post-nasal adenoids (§ 128).

A great deal of information may be derived by passing the finger
behind the soft palate, but for this purpose it is generally necessary to
spray the pharynx with cocaine (10 per cent.). In young children, Pos-
terior Rhinoscopy is practically impossible. A digital examination may
be effected by introducing the forefinger (palmar surface directed towards
the posterior pharyngeal wall) and guiding it along the wall up to the
roof of the pharynx. If skilfully done, this causes nothing more than
an unpleasant surprise and is not resented by the little patient.

Our *first* inquiries concerning any given case of suspected disease of
the nose should be relative to the LEADING SYMPTOM, especially whether
there be any nasal discharge, and whether it is inodorous or foul smelling.
We cannot depend upon the patient's statement on this point, because
very often the same disease which causes a foul discharge may blunt the
sense of smell. *Secondly*, we must investigate the HISTORY, and whether
any of the other symptoms above mentioned were present. *Thirdly*, we
must proceed to the PHYSICAL EXAMINATION by testing whether the patient
can breathe freely through each nostril separately ; by examining the
anterior, and, if necessary, the posterior nares.

Classification.—Diseases of the nose, like those of the throat, are best
classified by the PHYSICAL SIGNS met with on examination—viz., **nasal
discharge, nasal obstruction, epistaxis**—and their **causes.**

(*a*) ACUTE INODOROUS DISCHARGES (Acute Rhinorrhœa)—the causes
of which are—

I. Acute Rhinitis ; II. Syphilis (snuffles) ; III. Diphtheria, and other fevers ;
 IV. Coryza ; V. Hay Fever ; VI. Glanders.

(*b*) CHRONIC INODOROUS DISCHARGES (Chronic Rhinorrhœa)—the
causes of which are—

I Chronic Simple Rhinitis ; II. Chronic Hypertrophic Rhinitis ; III. Cerebro-

spinal Rhinorrhœa ; IV. Ulcerations of the Nose, Polypi, and occasionally Catarrh of the Sinuses.

(c) CHRONIC OFFENSIVE DISCHARGES (Ozæna), which have for causes—

I. Ulcerations and Bone Disease—Syphilis, Tubercle, and Lupus ; II. Atrophic Rhinitis ; III. Empyema of Antrum and other Sinuses ; IV. New growths and polypi breaking down, and impacted foreign body.

(d) NASAL OBSTRUCTION (Snoring and mouth-breathing)—the causes of which are—

I. Pharyngeal Adenoids ; II. Polypi ; III. Deviated Septum ;   IV: Hypertrophy of Turbinate ; and V. Foreign body and neoplasms i.. adjacent parts.

(e) EPISTAXIS, the causes of which may be Local or General.

§ 153. Acute (or recent) **Inodorous Discharge from the Nose (Rhinorrhœa).**—*Discharge is a frequent symptom when disease of the nose is present, and we should endeavour to ascertain if this be odourless or offensive, although these are, of course, only relative terms, and the two groups cannot be sharply defined. Among the causes of* ACUTE INODOROUS DISCHARGE, *congenital* SYPHILIS *should be suspected in infancy;* DIPHTHERIA *in childhood;* CORYZA *in adults.*

I. **Acute Rhinitis** may be set up by *irritation* of any kind, as the vapour or dust of some trade, or by any injury.   For instance, a profuse discharge from one nostril in a child should always make us suspicious of his having inserted a pea, marble, or other *foreign body*, although the history may be wanting.   But its commonest cause is a " cold " (see Acute Coryza below).

II. **" The Snuffles."**—In infants a few weeks old, congenital syphilis is almost invariably attended by a profuse nasal catarrh, known familiarly as the " Snuffles." Syphilitic Snuffles is obvious in the presence of a purulent rhinitis and other associated symptoms.   There is a non-syphilitic form occurring at birth, in which the nose is relatively dry and free from rhinitis.   This form is due to rigidity of the soft palate, and may be cured by placing the index finger behind the palate and gradually pulling it forward, thereby loosening it.

III. **Diphtheria** and other fevers.   A profuse nasal discharge excoriating the upper lip, with slight elevation of temperature, and prostration, coming on suddenly in a child or young person previously healthy, is so characteristic of diphtheria that the disease may almost be diagnosed from these features alone.

IV. In **Acute Coryza,** " catarrh," or " cold in the head," there is profuse muco-purulent discharge attended by sneezing, running from the eyes, and febrile symptoms with frontal headache, extending over a few days. It is usually attributed to some exposure to cold (" a chill ") ; but it frequently prevails in an epidemic form, and is then of microbic origin. It is predisposed to by cold and damp weather, by adenoids, and the other causes of chronic rhinitis.   It is not a serious disorder, but its

repeated occurrence may lead to middle-ear catarrh, or to bronchitis by extension.

Treatment of " Catarrh."—In severe cases it is advisable for the patient to keep in bed. At the outset a full dose of Dover's powder given at night, or a mixture of tr. aconiti ℥ i. (0·06), liquor ammoniæ acetatis, with other salines, every two hours, may cut short the disease. Locally, sprays of cocaine (2 to 4 per cent., applied with caution, occasionally), or equal parts of boracic acid and borax dissolved in water, or camphor and menthol (1 in 60 of paroleine), may abort the disease. Ferrier's snuff [1] is also useful. Inhalations of camphor, menthol, or vinegar taken at night are reputed to be efficacious. Vaccines are efficacious in many cases. An autogenous vaccine can be used to abort a threatening coryza.

V. **Hay Fever,** or Hay Asthma, is a severe catarrh of the nasal mucous membrane and conjectivæ, coming on fairly regularly in the summer or autumn of each year, connected with the inhalation of the pollen of flowers. It is accompanied by the symptoms of severe coryza which come on somewhat suddenly in a person predisposed, who has been outdoors, and are attended by a certain amount of constitutional disturbance. There appear to be two clinical varieties of this disease—(i.) where the symptoms are chiefly constitutional ; and (ii.) where the symptoms are chiefly local. In the latter there is generally hypertrophy of the inferior turbinate, which constitutes an important predisposing factor.

Etiology.—The disease is an exudative catarrh produced in hypersensitive persons by the anaphylactic action of certain pollens. The normal digestive action of the nasal mucous membrane on the pollen fails ; a foreign protein therefore enters and causes the anaphylactic phenomena. Some people cannot go within a mile of a hayfield without developing the disease. It is *diagnosed* from simple coryza chiefly by its seasonable occurrence. It resembles asthma especially in its periodicity, but differs in that the nasal, instead of the bronchial, mucous membrane is involved. The malady is not a fatal one, but causes serious discomfort and inconvenience. Sometimes people get rid of it as they get older, but in others it continues throughout life.

Treatment.—The first indication is the avoidance of the cause. This may be accomplished by a sea-voyage, residence at the seaside at a high altitude, or by living indoors in the city, taking care that no plants or flowers enter the house. But there is no rule in this respect ; for some do better at a high altitude, others at a low one ; some get better at the seaside, others in a town. Sod. Bicarb. ℈i. (4) thrice daily often does good, and in some cases large doses of calcium chloride are successful. If the disease extends to the bronchi, asthma papers and cigarettes should be employed. Locally, means should be taken to prevent the pollen reaching the mucous membrane. For this purpose Brunton recommends smearing the nostrils with zinc oxide ointment, which, by remaining longer unmelted, is more efficacious than other ointments. Antiseptic sprays destroy the pollen. Of these quinine, ¼ grain to the ounce, dissolved in normal saline solution, as being less irritating than water, gives good results. Sir Andrew Clark recommended swabbing out with hyd. perchlor., gr. ℔. (0·06) and quin. hydrochlor., gr. ℥.(0·13) in glyc. ac. carbol. To relieve the discomfort, cocaine tabloids, ¼ grain (0·016), inserted in the nose, sprays of cocaine (4 per cent.), or menthol (20 per cent.) are used. The mucous membrane, if thickened, must be treated as in hypertrophic rhinitis. Pollantin brings about a degree of passive immunisation ; vaccine treatment is given to cause active immunity, and must be started early in the year. Ionisation of the nasal mucosa by direct application of pledgets of cotton

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[1] Bismuth Subnit., ℥vi. (24) ; Morph. Hydrochlor., gr. ii. (0·13) ; Pulv. Acac., ℥ii. (8).

wool soaked in a 1 per cent. solution of zinc chloride, prevents the onset of an anticipated attack.

VI. **Glanders.**—The copious discharge of viscid semi-purulent matter from the nostrils is one of the earliest symptoms of Farcy, or Chronic Glanders (§ 389).

VII. **Myiasis** is chiefly met with in tropical countries. It is due to the presence of maggots. The eggs from which they hatch are laid by a fly on the nasal mucous membrane, usually while the patient is asleep. Inhalation or local application of pure chloroform is the usual remedy, but insufflations of calomel are also successful.

§ 154. In **Chronic Nasal Discharges** *it is still more difficult to draw the line between odorous and inodorous discharges, since many of the conditions, though odourless at the outset, become offensive later on, and it will generally be necessary to pass in review all the conditions mentioned in this section and § 155 below.* The following are the chief causes of INODOROUS DISCHARGE :

I. **Chronic Rhinitis** is a chronic inflammatory condition of the mucous membrane of the nose, attended by increased secretion, and usually by thickening. It occurs in three forms : (a) SIMPLE ; (b) HYPERTROPHIC (*infra*) ; (c) ATROPHIC (§ 155). The first two give rise to an *inodorous*, but the ATROPHIC to an *odorous* discharge.

CHRONIC SIMPLE RHINITIS consists of a chronic congested, and sometimes, later on, a hypertrophied state of the mucous lining of the nose, attended by a continuous mucous or muco-purulent discharge. There is generally some nasal obstruction, giving rise to altered voice and snoring.

Etiology.—(i.) It is predisposed to by cardiac and pulmonary disease, alcoholism, and the strumous diathesis. It may be *determined* by (ii.) recurrent attacks of neglected coryza over a long period of time ; (iii.) the injury caused by an unsuspected foreign body, in which case the condition is generally confined to one side ; or (iv.) the constant irritation of dust and noxious vapours—*e.g.*, in masons, fustian-cutters. (v.) It is often associated with adenoids, enlarged tonsils, a deflected septum, and other causes of obstruction in the nose. (vi.) Recurrent rhinitis may be·due to obscure antral or sinus trouble.

Prognosis.—The disease is chronic, and requires prolonged treatment. The chief fear is that middle-ear catarrh may result from the extension of the inflammation up the Eustachian tube. Even apart from this, it is very important to treat these cases in strumous children, because the condition interferes with the respiratory functions of the body.

Treatment.—In the early stages alkaline washes—bicarbonate of sodium, gr. xv. (1), and borax, gr. v. (0·3), or carbolic acid, gr. iii. (0·2) to ʒi. (32)—sniffed up or given by the nasal douche. This is followed later on by a spray of menthol and eucalyptol (gr. xxx. (2) to ʒi. (32) of aquol or paroleine), or an ointment of cocaine and thymol (gr. x. (0·6) to ʒi. (32) of white vaseline), or by the use of the ammonium chloride inhaler. Constitutional treatment is necessary, by means of tonics, cod-liver oil, and malt. Alcohol should be avoided, and a high and dry climate should be sought. In the later stages, the only satisfactory method of treatment

is applying chronic acid (1 or 2 per cent.), or, still better, the galvano-cautery.

II. **Chronic Hypertrophic Rhinitis** is a special form distinguished from the preceding by the fact that there is considerable hyperplasia of the nasal mucous membrane, especially over the inferior turbinate bone at its anterior and posterior ends. It presents the same symptoms as the preceding, but in a greater degree. Even in slight cases it is apt to be accompanied by headache and mental depression. It is frequently associated with adenoids. The *Prognosis* is on the whole less favourable. The *Treatment* is much the same, but more active measures are indicated, and especially treatment by the thermo-cautery.

III. **Cerebro-spinal Rhinorrhœa** is a continual dripping of a watery, clear fluid (cerebro-spinal fluid) from the nose, due to the formation after injury or disease of a communication between the nasal cavity and the sub-arachnoid space. The fluid passes through the cribriform plate of the ethmoid. Its nature is at once recognised by the fact that it reduces Fehling's solution. Little can be done for the condition; interference is apt to be followed by meningitis. The flow sometimes ceases spontaneously. Some cases have been successfully treated by applying to the nasal mucosa irritants which cause swelling and occlusion of the lumen of the sinus.

IV. **Ulcerations of the Nose, Polypi, Disease of the Sinuses,** occasionally produce inodorous discharges, but the discharge is more often offensive (see below). CHRONIC FRONTAL SINUS EMPYEMA is, however, attended by (i.) a purulent, *non-fœtid* nasal discharge, (ii.) frontal or supra-orbital headache or feelings of discomfort, and (iii.) more or less well-marked nasal obstruction, caused by inflammatory enlargement of the middle turbinate body, or by polypi. Headache only occurs from retention, and not when drainage is free. There may be tenderness on pressure over the affected side.

V. **Post-nasal Catarrh** is a condition in which the catarrhal processes are confined to the naso-pharynx. Its importance is derived from the fact that it is not easily recognised unless looked for. The constant swallowing and absorption of septic matter from the post-nasal focus may give rise to serious constitutional results, and by direct extension the ear or sinuses may be affected. Treatment is on the same lines as that for the commoner forms of nasal catarrh.

§ 155. Ozæna or a **Chronic Offensive Discharge** *from the nose may occur in the later stages of* MANY OF THE CONDITIONS *mentioned in the preceding section. But the chief causes of foul discharge from the nose are as follows :* the commonest and foulest occurring in ATROPHIC RHINITIS *in the young ;* SYPHILITIC DISEASE *in middle life ; and* CANCER *in the aged.*

Foreign bodies *(which have already been referred to) and* Polypi, *both of which may cause one-sided ozæna, are described under Nasal Obstruction* (§ 156), *which is their leading symptom. It will be necessary to give some detailed account of*—Ulcerations and Bone disease; Atrophic Rhinitis ; and Empyema of the Sinuses.

I. **Ulcerations and Bone Disease** attacking the nose are mostly of syphilitic, occasionally of tuberculous, origin. Neoplasms in the later stages ulcerate, but in the earlier stages give rise to Rhinitis or Nasal Obstruction.

(a) **Syphilitic Rhinitis.**—In the early stages of syphilitic infection we may get an acute catarrh with superficial ulceration, which is the condition

found in children with congenital syphilis, known as " snuffles." In the later stages gummata form in various situations, which *rapidly involve the bone* and other parts ; the discharge then becomes very foul. The ulcers have the same character as those affecting the throat (*q.v.*). There is a positive Wassermann reaction. ¹

(*b*) **Tuberculous Ulceration** more often involves that part of the nose near the orifice, but otherwise the ulcers much resemble the preceding. They are differentiated from them by their very much slower progress, as well as by their site. The bones are rarely attacked, and consequently the discharge may be more or less inodorous ; and there is rarely the falling in of the bridge of the nose, which so frequently occurs in tertiary syphilis. The Wassermann reaction is negative. The ulceration of *Lupus* differs but little from the true tuberculous ulceration, except that lupus vulgaris usually involves also the skin of the alæ nasi, *whence it has probably spread.*

Atrophic rhinitis is distinguished from these ulcerations by the pallor and thinning of the mucous membrane, the absence of visible ulcers, and the absence of a history of evidences of syphilis or tubercle respectively.

The *Prognosis* of nasal ulceration is fairly good if the patient come under treatment early, otherwise it leads to considerable destruction of tissue. Tuberculous ulceration may slowly lead to the destruction of the *alæ* of the nose, but syphilis results in the most extensive destruction of the *bones* both of the septum and the palate ; the bridge of the nose falls in, and the anterior nares may be represented by a single gaping orifice. It is this extensive and rapid destruction which is so pathognomonic of nasal syphilis.

The *Treatment* should be much more prompt and vigorous in ulceration of the nose than in chronic rhinitis and similar affections, because of the destruction which ensues. Carbolic and astringent sprays are useful palliatives, but surgical measures are called for if the bone is involved. All dead bone must be removed. Tuberculous ulcers be scraped. Salvarsan or large doses of potassium iodide lead to rapid healing of syphilitic ulcerations.

II. **Atrophic Rhinitis,** also known as idiopathic or true ozæna, is characterised by (i.) a thick, foul discharge, which is sometimes profuse, sometimes scanty ; (ii.) the nasal cavities are often large, and the bridge of the nose broad and sometimes depressed. The mucous membrane is thin, pale, and covered with crusts, hard, adherent, and decomposing. Sometimes it is unilateral—*e.g.,* in cases of deviated septum. A certain amount of chronic pharyngitis is usually present. (iii.) The breath has a foul odour,·which is not detected by the patient, as the sense of smell is blunted. It is *Diagnosed* from the other causes of ozæna by the absence of ulceration, the presence of atrophied mucous membranes, and wide cavities.

Etiology.—(i.) It is commoner in the young and in women. It usually starts before sixteen years of age. (ii.) Unilateral atrophic rhinitis is mostly due to some local cause, such as deviated septum or sinus disease, the narrower side being healthy. (iii.) The exciting causes of bilateral atrophic rhinitis are obscure : it has been said to follow chronic rhinitis in strumous children ; (iv.) in some cases it is a sequence of hypertrophic rhinitis.

Prognosis.—Prolonged treatment is necessary for its cure, and even this is not very hopeful if the disease be advanced. The disorder is generally most marked at about twenty years of age ; it becomes less troublesome at middle age, and, as it

gradually disappears with advancing years, we may presume that it tends slowly to spontaneous cure.

Treatment.—Alkaline and antiseptic douches and sprays are indicated, as in § 154. To stimulate the mucous membrane, nasal tampons of cotton wool, soaked in glycerine, are used. These are useful in unilateral rhinitis, as they insure respiration through the narrower cavity. The nose may be swabbed out with silver nitrate (1 in 50), or with trichloracetic acid (5 to 20 parts in 1,000), which removes the smell. Constitutional treatment is also advisable. Vaccines assist certain cases.

III. **Chronic Empyema of the Antrum** and other sinuses is a term applied to a chronic suppurative inflammation of the lining membrane, though the term " empyema" should be reserved for cases in which there is retention. The most constant and cardinal symptom is a purulent or sero-purulent discharge from *one nostril,* which is generally offensive or sickly. It may arise as an extension of nasal catarrh, or various suppurative nasal conditions (syphilis), tubercle, bone disease, etc.

Empyema of the Antrum may be due to irritation from a tooth. Many of the patients have had decayed teeth in the upper jaw. It is recognised by the discharge being intermittent, returning usually about the same time each day, and flowing freely when the head lies on the opposite side, or is lowered between the knees. The discharge may be seen coming from beneath the middle turbinate. If a bright light is held in the mouth, the cheek of the affected side remains darker than the other (transillumination).

Discharge from the *frontal* or *anterior ethmoidal* sinuses flows best when the patient is upright. It comes from under the middle turbinate, and there is often pain in the brow and orbit (see also p. 227).

Discharge from the *posterior ethmoidal* and *sphenoidal* sinuses flows over the middle turbinate and down into the pharynx. There may be exophthalmos, ptosis, strabismus, etc., with disease in this locality.

Various constitutional symptoms are recognised as being associated with sinus disease, due probably to the toxæmia which results from septic absorption. Lassitude, headache, occasional elevations of temperature, and numerous nervous and vasomotor symptoms are amongst the commonest. They generally present a periodic or paroxysmal character. Trifacial neuralgia may also result from sinus disease. If overlooked or neglected, empyema may excite middle-ear catarrh (with tinnitus, deafness, etc.), recurrent nasal catarrh, and nasal polypi.

Prognosis and Treatment.—Sinus empyema is chronic and intractable, but very rarely fatal. The treatment is based on surgical principles, but the chief indications are free drainage and stimulation of the chronic inflammation until it takes on a more healthy action of repair.

IV. **Neoplasms and Polypi** (§ 156), and **Impacted Foreign Body** (§ 153, I.), are referred to elsewhere.

§ **156. Nasal Obstruction, Snoring, and Mouth-breathing.**—*Nasal obstruction may be partial or complete, and it may exist on one or both sides. It is met with in a greater or less degree in nearly all of the various nasal conditions previously discussed, and it is a marked feature in* HYPERTROPHIC RHINITIS (p. 227). *Its commonest cause in children is* PHARYNGEAL ADENOIDS (§ 128). *It is also a cardinal symptom in* NASAL POLYPI, DEVIATION OR SPUR OF THE SEPTUM, ALAR COLLAPSE, FOREIGN BODIES, NEOPLASMS, *and* ABSCESSES.

Effects.—Apart from the inconvenience of snoring, nasal obstruction renders the individual prone to pharyngitis, stomatitis, bronchial catarrh, and other consequences due to the entry of cold air into the lungs without being properly warmed by its passage through the nose. Among the other consequences are a nasal quality of the voice, distortion of the chest

(when arising early in life), and impeded respiratory functions of the body generally. These disorders consequently assume an importance quite out of proportion to the degree of local mischief.

I. **Pharyngeal Adenoids** are of very frequent occurrence. They constitute one of the forms of granular pharyngitis, and the disease has been referred to under that condition (§ 128). It is the most frequent cause of mouth-breathing and snoring in children. It is often overlooked by parents, a circumstance greatly to be regretted for three reasons. In the first place, it is one of the most potent causes of chronic otitis media and deafness in after-life ; secondly, it impairs the respiratory functions of the body, as just mentioned ; and thirdly, the open mouth and vacant aspect, which are so characteristic, produce an appearance of backward intelligence which in point of fact often results.

II. **Polypi,** or pedunculated tumours, are the most frequent new growths in the nose. Polypi are of three kinds : (a) GELATINOUS ; (b) FIBROUS ; and (c) MALIGNANT.

(a) GELATINOUS or MUCOUS POLYPI are the most common form of polypi. They usually consist of myxomatous tissue, believed by some to be associated with inflammatory disease of the subjacent bone. They are often multiple, and most often grow from the muco-periosteum of the upper and middle turbinated bones. Their detection is not difficult, for in addition to the feeling of " stuffiness " and the watery discharge (which may be intermittent), they are easily seen through the nasal speculum as pale grey glistening bodies. They are apt to recur after removal, but are not malignant in other respects.

(b) FIBROUS POLYPI grow from the roof of the naso-pharynx. By their growth they displace the parts around and are apt to give rise to " frog face." The discharge is often foul, and may be hæmorrhagic. They sometimes become malignant (fibro-sarcoma). They may occur at any age.

(c) MALIGNANT POLYPI may be either fibro-sarcomatous or carcinomatous. They are known by their rapid growth, and the resulting deformity of the face, " frog-face," and by the offensive and hæmorrhagic discharge. Sarcomatous growths are chiefly met with in the young ; carcinomatous in the aged.

Prognosis and Treatment.—The benign polypi are not dangerous to life, but are liable to recur. Malignant growths give rise to a condition of considerable gravity. Occasionally fibrous tumours atrophy. Gelatinous polypi, arising as they do from the anterior part of the cavity, can generally be removed by means of a nasal snare or forceps ; but the other varieties, springing usually from the posterior parts and infiltrating the tissues around, may require an operation of some magnitude.

III. **Deviated Septum and Nasal Spur.**—The nasal septum is rarely quite in the median line, but the displacement is often considerable. Sometimes it results from injury. Various consequences may ensue, such as hypertrophied turbinate on one

side, atrophic rhinitis on the other.   When an angle is formed in the septum nasi, it
is spoken of as a " spur," and this is most readily dealt with by the surgeon.

IV. **Hypertrophied Turbinate** is met with usually either as part of, or a consequence
of, chronic hypertrophic rhinitis.   It may occur on one or both sides, and in either
case, in narrow nostrils, produces partial obstruction, snoring, and mouth-breathing.
It is removable by turbinectomy ; sometimes the thermo-cautery is employed (see
Hypertrophic Rhinitis, p. 227).

V. **Foreign Bodies** within the nose, **Neoplasms**, and **Abscesses** in adjacent parts
may also produce *unilateral* nasal obstruction.

§ 157. Epistaxis (bleeding from the nose) may be a symptom of nasal
disorders, but if in any appreciable quantity it is usually an evidence of
some general disorder.   Not infrequently both general and local causes
are in operation, and the *nasal cavities should always be carefully examined.*
The blood-vessels give way in this situation (sometimes as a kind of safety
valve) merely because they are thin-walled, numerous, and near the sur-
face.   So much is this the case that the diminished atmospheric pressure
to which mountaineers are subjected is sufficient to produce nose bleeding
when they reach great heights.   The *Causes* may be divided into two
groups—Local and Constitutional.

   (*a*) LOCAL CAUSES, in which the hæmorrhage consists usually of little
more than streaks, may arise from any marked congestion of the mucous
membranes, such as that which accompanies adenoids, acute rhinitis,
worms in the nose ; [1] or as a consequence of mechanical violence, applied
either directly to the nose or to the base of the skull.   Any serious destruc-
tive disorder—such as new growths, especially malignant, syphilitic,
tuberculous, or other ulcerations (which if small are *very apt to be over-
looked*)—may be attended by a certain amount of recurrent bleeding.   In
these circumstances the hæmorrhage is usually an intermittent and sub-
ordinate feature.   The diagnosis rests on the characters already given.
When small in quantity the blood often passes backwards into the throat
and is swallowed, or it may be expectorated or coughed up, and be
mistaken for hæmatemesis or hæmoptysis.

   (*b*) With CONSTITUTIONAL CAUSES the bleeding is usually, although
not always, of larger quantity, and it may, indeed, be so profuse as to
endanger life.   The blood in this group comes from a spot near the anterior
part of the septum.   Among the *predisposing causes* none is more frequent
than an idiopathic tendency which exists in certain individuals to bleed
upon slight provocation, a tendency which runs in families.   Without
amounting to hæmophilia, certain persons undoubtedly present some
inherent quality which renders them more liable to bleed from their
mucous surfaces, with or without a wound.   It may exist in only one
member of a family, but more often in several brothers and sisters.   I
have often noticed that such a predisposition may exhibit the pheno-
menon of atavism and skip a generation.   Epistaxis is more frequent in

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[1] Dr. Manasseh relates the case of a child with epistaxis in whom a leech was
found in the nose.   This had gained entrance by the child drinking at springs in a
district where leeches abounded in the water.

children, especially in boys. It is also met with in the aged, but only when vascular disease and some of the other conditions about to be mentioned exist. The constitutional causes may be grouped under (a) *Alterations in the Cardio-vascular System*, and (b) *Altered Blood States*,

(a) Epistaxis occurring for the first time in an apparently healthy person over forty years of age should always give rise to the suspicion of chronic Bright's disease. It affords us, moreover, an indication for the treatment of this malady, of which advantage may sometimes be taken, for it relieves the vascular tension which would otherwise seek relief in some less favourable situation. For instance, I have observed several patients who, after repeated admissions to hospital for epistaxis, have finally come in to die of cerebral hæmorrhage. Epistaxis is a frequent consequence of cardiac valvular disease, emphysema, chronic bronchitis, and cirrhosis of the liver. It may also be an evidence of lardaceous or other disease of the vessels. Finally, epistaxis is one of the forms of vicarious menstruation, and, like the bleeding which may take place in hysteria and other conditions where the vaso-motor system is disordered, we must regard this as an extreme effect of disease of the sympathetic nervous system.

(b) Concerning *Altered Blood States*, it may occur with purpura, hæmophilia, scurvy, leukæmia, anæmia (simple, and especially pernicious), and the specific fevers, especially typhoid, rheumatism, and the hæmorrhagic forms of the exanthemata. It is in children a not infrequent prodromal manifestation of whooping-cough and similar microbic disorders.

Prognosis.—Slight epistaxis in children is of no consequence, but when occurring for the first time in persons at or past middle life it should receive our serious attention, and its cause should be carefully investigated. Inquiry should always be made as to whether it has occurred previously in the life of the individual, because, as above mentioned, certain persons have this tendency, and in these the symptom may not be of much importance.

Treatment.—The indications are—first, to check the hæmorrhage if profuse; and secondly, to ascertain the cause.

The epistaxis which accompanies Bright's disease, and the congestion of cardiac and pulmonary disease should not be checked unless the amount be profuse. In such cases the epistaxis is usually preceded by headache, and is accompanied by high blood-pressure. It may be one of Nature's methods for the relief of congestion, as evidenced by the fact that the headache and the high blood-pressure are relieved by the hæmorrhage. In all cases of epistaxis, the first thing to do is to examine the blood pressure. So long as this remains high or moderate no harm can accrue from the epistaxis.

(a) The treatment of *the attack* resolves itself into checking the hæmorrhage. The patient should be kept perfectly quiet, with the head erect, and chin forward, the head being cool, the feet warm, with hot bottles if necessary. The arms may be raised above the head and ice applied

to the lower cervical spine. A homely substitute for the latter has long been in vogue in the form of the front-door key. Pressure should be kept up over the anterior part of the septum by the thumb and forefinger externally. The cautery applied to the bleeding spot is also efficacious. Other useful measures consist of the application of adrenalin to the site of the hæmorrhage, if this can be discovered, or the use of styptic sprays of hammamelis, catechu, vinegar, lemon-juice, etc. Finally, if all these fail, the posterior nares must be plugged. If hæmorrhage continues for several days, internal treatment must be given—calcium chloride and ol. terebene ℥xx (1·3) in tragacanth every four hours. Serum, especially horse serum, 10 to 20 c.c. daily, may be injected subcutaneously.

(b) *Between the attacks* a very *thorough investigation* of the nasal and post-nasal cavities must be made. Minute lesions, quite sufficient to cause epistaxis, are very easily overlooked. The treatment of recurrent epistaxis is not always an easy matter, for the cause is often obscure, and we are often driven to regard the case as belonging to the idiopathic group above referred to. In a good many cases iron is efficacious in warding off the attacks ; and calcium chloride by increasing the coagulability of the blood.

THE THYROID GLAND

This gland is anatomically connected with the upper respiratory passages, but is physiologically quite separate. It supplies an internal secretion which is necessary to the well-being of the individual, and it is in close relationship with the other ductless glands, the supra-renal, the pituitary, the pancreas, the ovary and others. In health it enlarges at puberty, during menstruation, sexual excitement, pregnancy, lactation, and in the presence of most acute specific fevers, notably rheumatic fever.

Symptomatology.—There are two opposite clinical conditions which may arise from disorder of the thyroid gland. In one there is a *diminished* thyroid action, a condition of *Athyroidism*, the symptoms of which (lethargy, lowered vitality, and impaired growth and development) are similar in kind but less in degree to those of Myxœdema and Cretinism. The other condition is one of *increased* (or perverted) thyroid action or *Thyroidism*, the symptoms of which resemble Graves' disease ; and these, with the exception of the proptosis, can be produced by the internal administration of thyroid gland or extract in large doses. It is important to remember that the size of the gland is not in any degree a guide to which of these two sets of symptoms are to be expected in a given case, for enlargement of the gland is consistent with atrophy of the glandular elements and diminution of function ; while what appears to be a small gland may be functionally very active.

§ 158. Physical Examination and Classification.—There are but two physical signs referable to the thyroid gland—viz., enlargement or decrease of volume and consistency. When the alteration of volume is only slight it is

difficult, if not impossible, to estimate it with accuracy, because it is partially covered by muscles, and is intimately connected with the trachea and other deeper structures. The patient should be instructed to let his head fall forwards and *to swallow* whilst we endeavour to palpate the gland. The thyroid rises during deglutition as no other neck tumour or organ does. Some idea may be obtained of the progress of a case by measuring the neck from time to time, always exactly at the same level.

When the thyroid fails to develop normally, part of the thyroid tissue may be left at the base of the tongue. There it forms a painless, soft swelling in the mid-line, which may not attract notice until it enlarges at puberty or later. If the swelling be removed, myxœdema or hypothyroidism will follow ; if the physician realise the nature of the swelling, this error will be avoided.

Classification.—In general, *enlargement* is attended by a condition of thyroidism (*e.g.*, Graves' disease), and a *diminution* by a condition of athyroidism (*e.g.*, myxœdema) ; and there are two well-marked types of disease which are *usually* associated with enlargement, and two with diminution in volume of the thyroid gland.

(*a*) The two diseases (besides cancer and other neoplasms) in which **enlargement of the thyroid** is—at some stage of the malady—the essential or pathognomonic feature [1] are—

I. GRAVES' DISEASE—or Exophthalmic Goitre—is the term applied to that form of enlargement of the thyroid which is attended by proptosis, and by numerous cardio-vascular and nervous symptoms, with marked disturbance of the general health. These general symptoms collectively constitute THYROIDISM. They are often present long before there is any visible enlargement of the gland.

II. SIMPLE GOITRE or "BRONCHOCELE" is the term applied to a simple increase in size of the thyroid gland, either congenital or coming on in childhood or early adult life, generally attended by slight though definite diminution of function.

ENLARGEMENT OF THE THYROID is also met with in (i.) chlorosis, and (ii.) in the acute specific fevers, especially rheumatic fever. It may go on to abscess formation, as in typhoid fever. (iii.) Rarely, it enlarges during the course of leukæmia. (iv.) Acute hæmorrhage may occur into the gland. (v.) The thyroid may enlarge with iodism. (v.) Chronic inflammation or "ligneous thyroiditis" leads to the slow formation of a hard mass of fibrous tissue which may involve surrounding structures, and become dangerous to life from pressure upon the trachea ; surgical treatment may be required.

(*b*) There are also two diseases in which **atrophy of the thyroid**—or at any rate a diminution of its function (and usually of its size)—is the essential feature of the malady :

I. CRETINISM is the term applied to the condition of stunted growth

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[1] In ACROMEGALY (Chapter XVII) the thyroid is sometimes slightly enlarged or diminished in size, but it has generally been regarded as a subordinate feature in this rare and strange disease. In some cases of CRETINISM (which is included in group *b*) the thyroid gland is considerably enlarged, but *deficient* thyroid action constitutes the chief factor of the disease.

(both in mind and body) of the individual, due to congenital atrophy or absence of the gland function, although the gland itself may be either enlarged or diminished in size. It is a condition of congenital ATHYROIDISM. The disease is endemic in certain districts.

II. MYXŒDEMA is the term applied to the group of symptoms (lethargy, low vitality, etc.) which, coming on in adult life, especially towards middle age, accompany *atrophy* of the thyroid gland. This is a condition of acquired ATHYDROIDISM.

It therefore follows that :

1. Increased or disordered thyroid secretion gives rise to profound disturbance of the general health, and neuro-vascular irritation (Graves' disease).

2. An innocent enlargement of the thyroid, unaccompanied by increased or disordered thyroid secretion, has no effect on the economy (as in most cases of bronchocele).

3. Simple absence or diminution of the thyroid secretion results (a) when it is congenital or comes on in early life, in deficient development, mental and physical (i.e., cretinism) ; and (b) when it supervenes in adult life, in lethargy and deficient vitality (myxœdema).

Diseases usually attended by thyroid ENLARGEMENT—*viz.*, GRAVES' DISEASE *and* BRONCHOCELE.

§ 159. Graves' Disease (Synon. : Exophthalmic Goitre, Basedow's disease) has been defined on the preceding page. Usually the onset is very insidious. There are *five* groups of symptoms, and the varieties of the disease depend on which of these predominate.

Symptoms.—(1) *Cardio-vascular* disturbances are among the earliest and most important symptoms. They are never absent, and may exist for months before any other evidence appears : (i.) Palpitation. (ii.) The increased frequency of the heart is accompanied by a rapid and sometimes feeble pulse, up to 150 or more on the slightest exertion or emotion. The rate may sometimes be reduced by absolute rest by 40 or 50. (iii.) Paroxysmal dyspnœa and a distressing sense of suffocation, produced and relieved by the same means as the preceding. (iv.) Evidences of cardiac disease, such as the murmur of dilatation is frequently present and in severe cases auricular fibrillation. (v.) Sometimes slight dropsy and albuminuria are observed.

(2) *Nervous* disturbances are always present. They are very variable : thus (i.) there may be nervousness, irritability, insomnia, depression alternating with excitement, hysterical attacks, melancholy, or mania. (ii.) Hyperæsthesia, perverted sensations, neuralgic headache, vertigo, tinnitus aurium, and hallucinations of sight or hearing. (iii.) Other fairly common symptoms are fine and rapid vibratile tremors of the hands (always), or of the lips (seldom). (iv.) Vaso-motor disturbances of many different kinds, intolerance of heat, sudden perspirations and cutaneous disturbances such as pigmentation and loss of hair. Diarrhœa is a very common symptom.

(3) *Thyroid Enlargement* is always present at some stage of the disease, though it is rarely the first symptom noticed by the patient, probably

because there are no means of detecting slight enlargements. Therefore in the early stages we have to rely upon the other symptoms. The enlargement varies considerably in different cases, and is by no means proportionate to the other symptoms, because the symptoms depend more upon the histological element of the gland which is involved than the degree of enlargement (p. 233). Mechanical effects of thyroid enlargement may be present (see Bronchocele), and occasionally alteration in the voice from this cause is the first symptom noticed by the patient.

(4) *Exophthalmos* (proptosis or protrusion of the eyeballs) is present in a varying degree, though sometimes not until late in the disease (Fig. 2, § 10). It is best detected by seating the patient in a chair, standing behind him, and looking down his forehead. As a rule no changes can be detected in the fundi. Later on, ulceration of the cornea occasionally takes place, either from neuro-trophic causes or from deficient protection.

Four signs of Graves' disease referable to the eyes bear the names of different physicians. *Von Graefe's* sign is a condition in which the upper eyelid does not follow the eyeball when this makes a downward movement. *Mœbius's* sign is an insufficiency of convergence of the two eyes when looking at a near point. *Stellwag's* sign is an absence or deficiency of blinking as an involuntary act. *Abadie's* sign in this disorder is an involuntary twitching or spasm of the levator palpebræ superioris. All except the first are present only in advanced cases, and are not therefore of very great value in the diagnosis.

(5) The *general health* of the patient is always disturbed. Anæmia is in proportion to the severity of the other symptoms. Progressive weakness is always present and progressive emaciation is marked, owing to the increased rate of the basal metabolism. There is usually a lowered sugar tolerance. The *varieties* of the disease depend on which of these five groups of symptoms predominates.

Etiology.—(i.) Upwards of 95 per cent. of cases are females. (ii.) A large number are young adults between the ages of fifteen and thirty. (iii.) Locality has no known influence. (iv.) Heredity sometimes plays a part. The family often also show neuroses such as hysteria and insanity. (v.) Fright, anxiety, love affairs, and mental overwork are potent factors in determining the disease. (vi.) Toxæmia (alimentary, oral sepsis, etc.) undoubtedly aggravates, and may even cause the onset of the disease in many cases.

Diagnosis.—The cardinal symptoms are : (i.) thyroid enlargement, (ii.) proptosis, (iii.) rapid cardiac action, (iv.) fine tremors of the hands, and (v.) mental and emotional instability ; (vi.) increased basal metabolism. The latter is used to determine the diagnosis in obscure and early cases.

Prognosis.—The duration of the disease varies from some twelve months to many years. Two years may be considered an average duration. It may certainly shorten life, but many very severe cases have recovered under modern methods of treatment. The mortality has been variously stated as from 10 to 50 per cent. ; probably more modern statistics would give only from 5 to 10 per cent. If the duration be prolonged, the disease

will certainly leave its mark upon the cardio-vascular system. Progress may be judged by estimating the basal metabolism. The prognosis in severe cases is anxious in the direction of cardiac degeneration and mental instability. Those who recover often develop myxœdema in later years.

Treatment.—The early recognition of the disease is very important, for a great deal can be done in the early stages. Rest in bed is the prime essential. Freedom from fuss and worry is no less important. The patients are always *difficile*, but those about them should avoid thwarting or arguing with them. Of drugs, belladonna is sometimes successful. Bromide of quinine often succeeds admirably. In most cases iodine in small doses appears to supply the deficiency of the perverted secretion, and to hasten the cure. All sources of toxæmia must be patiently sought for and eliminated. Vaccines aid those in whom the toxins can be isolated. Sometimes success is obtained by means of extracts of some of the other internal secretory glands. Thymus gland has been used with success. Extirpation of the thyroid or division of the isthmus has been successfully adopted as a remedial measure, and is indicated when the gland is greatly enlarged, causing dyspnœa, or when medical treatment has failed after a fair trial. Excision of part of the diseased gland, under local anæsthesia, has also been successful in a large number of cases.

Galvanism (descending) is often useful, and small doses of X-rays have given good results in many cases. The discovery that the thyroid is a protective gland, which neutralises circulating toxins, has led to the trial of various sera. Thus Mœbius has injected a serum prepared from the blood of thyroidectomised sheep, and Murray a serum from rabbits, fed on increasing doses of thyroid extract. *Rodagen* and thyroidectin, the desiccated milk of dethyroidised goats, have given good results. As a rule thyroid does harm.

§ 160. **Bronchocele** (Simple Goitre) is another form of enlargement of the thyroid gland. It may affect the whole organ, or only one of its lobes, or the isthmus. *Anatomically*, the enlargement may be due chiefly to parenchymatous increase or to cystic enlargement. The enlargement may be so great that the organ amounts almost to the size of an infant's head.

The *Symptoms* which attend the disease are those due to the mechanical pressure of the tumour, and it is by the absence of the cardio-vascular, nervous, and other symptoms that this condition is distinguished from Graves' disease. The chief pressure symptoms are referable to the larynx and trachea. The voice is modified early in the disease, and vertigo, due to pressure on the vessels, may be present. The general health may be good, but the patient is usually somewhat anæmic, otherwise the symptoms are those of athyroidism rather than of thyroidism. It may be *Diagnosed* from other tumours in the neck by the fact that it invariably rises with the larynx during deglutition. The enlargement generally increases steadily, but it is only rarely that there is any danger from tracheal obstruction and asphyxia.

The *Etiology* of the condition is not well known. More often females are affected, and it sometimes starts during pregnancy, or a catamenial period, but it is most prone to start during adolescence. The disease is endemic in certain districts in England, America and on the Continent, especially Switzerland and the Tyrol. Recent work has proved that in these places the water is deficient in iodine. All cases however are not so simple. In some the assimilation of iodine is defective, possibly due to

intestinal conditions such as absence of vitamin in the diet, or excess of bacteria; in others it may be that the thyroid cannot utilise the iodine. McCarrison's work proved that goitre in Chitral and Gilgit was due to fæcal contamination of the drinking water. The work of McClendon and Hathaway showed that most forms of goitre were caused by deficiency of iodine, and they pointed out that when fish is canned its iodine content is lost. In ordinary diet iodine is obtained from milk, butter, fruits and leafy vegetables.

Treatment.—The water used for drinking purposes should be boiled. In McCarrison's cases cures were obtained by vaccines prepared from the stools, and by intestinal antiseptics, especially thymol and lactic acid bacilli. In this country it is found that small doses of thyroid extract, gr. ¼ to ½ (0·016–0·03), together with the local application of Ung. Pot. Iod. to the tumour will generally effect a cure. Indeed, iodine in small doses cures most cases. In certain parts of Switzerland, 1 to 2 grains of iodide of potassium were given weekly, sometimes in salt, with resulting cure and prevention of goitre. Surgical interference may be necessary.

Disease in which the thyroid is usually DIMINISHED *in size—viz.,* I. CRETINISM, II. MYXŒDEMA. *The latter is described elsewhere, since the leading symptom is General Debility (Chapter XVI).*

§ 161. **Cretinism** is a condition of dwarfism and deformity attended by mental imbecility, due to an absence or perversion of the thyroid secretion, and is endemic in certain districts. In advanced and typical cases the face is characteristically broad and flat, the tongue protrudes from the mouth, the eyes are wide apart, and the head is brachycephalic (*i.e.,* broad transversely). The skin and hair are dry and coarse, and the mental condition is extremely backward. In severe cases the body may be so dwarfed that a person of twenty is the size of a child of five. The limbs are shortened, the neck stunted; pads of fat are present above the clavicles; the hands are short and square (spade-like), the abdomen prominent and an umbilical hernia is often present. The thyroid may be enlarged, small, or absent (see Figs. 4A and 4B, § 19). Rushton Parker distinguishes three *varieties,* both etiologically and pathologically. In one, the thyroid is embryologically not developed, or only partially developed, the cause being presumably akin to that which brings about other embryological deficiencies, such as acardia, acephalia, etc. In a second, the thyroid undergoes the same changes as in endemic goitre, and doubtless from the same cause, any differences being due to loss of thyroid function. In a third, the thyroid, after performing its functions healthily for a time, atrophies, doubtless from causes akin to those of adult myxœdema.

Etiology.—Cretinism is endemic in certain districts, *e.g.,* the valleys of Switzerland. Cases occur also in certain parts of England, especially in the valleys of the Lake District and Derbyshire. Sporadic cases are found in healthy families. It is associated with deficient thyroidal function, and hence may be regarded as congenital myxœdema. In slight cases of cretinism the diagnosis from other forms of mental deficiency may be difficult; the condition of the skin and hair are valuable diagnostic features. In Switzerland it was observed that goitrous women gave birth to cretins; when the goitre was cured with iodine the subsequent children were healthy.

Prognosis.—The patient may grow up capable of doing light manual work, or may remain an idiot. Under treatment begun early, the child may recover completely, but in other cases, although the body is greatly improved the mind does not improve in proportion.

Treatment.—Thyroid extract, beginning with ½-grain (0·03) doses (5 grains of raw gland), causes a rapid and remarkable change. The skin becomes soft, the general conformation normal, and, if the treatment has not been too long delayed, the mind assumes its natural vigour. The patient must *continue* to take the thyroid all his life, or else he will relapse. A case showing the remarkable efficacy of this treatment is figured in § 19.

Complete myxœdema is described in detail elsewhere (§ 449). It should be remembered that there are degrees of thyroid insufficiency which, though falling short of typical cretinism or fully developed myxœdema, are nevertheless sufficient to account for many of the minor troubles for which patients seek advice. In childhood such deficiency should be suspected if adenoids or nocturnal enuresis are present. In adults, especially in women about the menopause, increase of weight, falling hair, intolerance of cold, muscular fatigue, a slow pulse, a dry skin with a tendency to chronic eruptions, are all suspicious features. In younger women premature greyness, and in men premature baldness, are also suggestive. Rarefaction amounting to complete absence of the outer two-thirds of the eyebrow is a fairly constant sign. The treatment is by thyroid extract in very small doses—$\frac{1}{8}$ to $\frac{1}{4}$ grain (0·008–0·016) three times daily. Large doses often aggravate the condition.[1]

[1] Dr. Leonard Williams, " Adenoids, Nocturnal Enuresis, and the Thyroid Gland ' (Bale and Sons) ; also " Thyroid Insufficiency," *Clinical Journal,* 1909.

· CHAPTER VIII

THE MOUTH, TONGUE, AND GULLET

The Mouth

(Lips, Breath, Saliva, Teeth, and Gums.)

WE often regret that we cannot investigate the internal organs more thoroughly, but how seldom do we avail ourselves of the instructive information afforded by a thorough examination of the mouth ? Many of the indications of syphilis, hereditary or acquired, may be so revealed ; several other constitutional conditions produce symptoms in this locality, such as anæmia and lead-poisoning ; and a good idea of the general condition of the patient can be obtained from a careful inspection of the tongue. Many of the disorders special to the mouth are comprised among the " causes " of stomatitis. For the diagnosis of these disorders it is necessary to make a thorough examination of the LIPS, the BREATH, the SALIVA, the TEETH, and the GUMS. We will consider the symptoms referable to these structures in that order.

§ 162. The Lips.—*Dryness* of the lips is often one of the most conspicuous evidences of indigestion, and it is a very useful one, because this disorder has so few physical signs to assist us. The lips are *pale* in anæmia, they are *cyanosed* in advanced bronchitis with dilated right heart, and in many other conditions (see Cyanosis, § 28). This cyanosis is especially marked in congenital heart disease. The hard chancre of syphilis may occur on the lip, § 434. In elderly men epithelioma may occur on the lip. *Fissures* around the lips are an almost infallible sign of syphilis, especially when surrounded by a reddened infiltration. This infiltration helps us to distinguish a syphilitic fissure from the " cracked lip " which is the only condition liable to be mistaken for syphilis. *Cracked lip occurs mostly* in nervous children who lick and bite their lips and are exposed to cold winds. It can generally be remedied by the application of some simple ointment, such as zinc ointment or cold cream, whereas the syphilitic fissures do not yield to this treatment. By pressing the corner of the mouth inwards and forwards when the patient opens it, we may often detect a mucous patch surrounding a syphilitic fissure inside the mouth. The *scars* left by syphilitic fissures are also a useful indication of a previous attack, or still more frequently of the patient having had congenital

manifestations. They are white and stellate. (See § 11 for other conditions.)

§ 163. **The Breath** should be normally quite free from any kind of odour. Offensiveness of the breath may arise from several sources : (1) A want of cleanliness *in the mouth*, particles of decomposing food, pyorrhœa, septic teeth and dental caries, may give rise to a very offensive odour of the breath. (2) Septic tonsils, and other throat maladies, dyspepsia, constipation, and other conditions of the *alimentary canal*, and the derangement of digestion in fevers. (3) Some *diseases of the nose ;* thus it always accompanies ozæna. (4) A large cavity in the *lungs*, especially if *bronchiectatic,* and gangrene of the lungs produce a putrid odour (§§ 121 to 123). The odour of bronchiectasis is characterised by being intermittent ; it comes on suddenly, lasts a day or two, and disappears gradually. (5) Certain general conditions are attended by a more or less characteristic odour of the breath. Thus, in *diabetes* it is sweet ; in acute *alcoholism* it is alcoholic or ethereal. In *uræmia* it is said to be urinous. (6) Certain *drugs* give rise to a very characteristic odour in the breath—*e.g.,* turpentine (a resinous odour), chloral (odour of chloroform), bismuth (odour of garlic), and opium (odour of the drug). Alcohol, ether, chloroform, and many other volatile substances are partly excreted by the breath.

§ 164. **The Saliva** may be *increased* (i.) in inflammation of the mouth ; (ii.) in chronic gastritis, and in some other gastric conditions, there may be such a profuse flow of saliva during the night that it gives rise to the impression that the patient, in the morning, is vomiting clear alkaline fluid (water-brash or pyrosis) ; (iii.) in pregnancy, and in mania, hydrophobia, and some other diseases ; (iv.) after the administration of mercury, pilocarpine, iodides, bitters, and, according to some, alkalies and acids. The saliva may appear to be increased, owing to defective swallowing, in bulbar paralysis, myasthenia gravis and other paralytic conditions ; and with sore throat or other causes of difficult swallowing. The saliva is *decreased* (i.) in certain febrile states, (ii.) in diabetes, (iii.) severe diarrhœa, (iv.) chronic Bright's disease, and (v.) during the administration of atropine or daturin. A condition known as " dry mouth " (xerostomia) has been described in which there is a constant deficiency of saliva. It is sometimes associated with affections of the salivary glands, such as calculus.

§ 165. **The Palate** may be " cleft " from childhood, otherwise a hole in this situation is practically always an evidence of past syphilis. The *soft* palate shares in the diseases of the fauces (§ 127). It is a favourite position for the membrane of diphtheria, which in this situation forms an important means of differentiating the disease from follicular tonsillitis, the exudation of which never affects the palate. The *hard* palate is sometimes involved in the diseases of the floor of the nose. A swelling may appear here in abscess of the antrum, or in abscess dependent on disease of the lateral incisor tooth. The latter is the commonest cause of swelling in this situation.

Thirst accompanies all febrile conditions and inflammatory conditions

of the gastric mucous membrane. It is met with also in diabetes, after various causes of loss of fluid, e.g., diarrhœa, perspiration, hæmorrhage, and vomiting, after a diet excessively salted, and with dyspepsia.

§ 166. **The Teeth** are subject to a certain amount of variation, even in health. The *average* dates of the eruption of the temporary and permanent teeth are as follows :

Temporary or " Milk " Teeth.	*Permanent Teeth.*
About 6th to 8th month, lower central incisors.	About 6th year, first molars.
	7th „ central incisors.
About 8th to 10th month, upper incisors.	8th „ lateral incisors.
	9th „ anterior bicuspid.
About 12th to 14th month, first molars.	10th „ posterior bicuspid.
About 18th to 20th month, canines.	11th to 12th year, canines.
About 2 to 2½ years, posterior molars.	12th to 13th „ second molars.
	17th to 25th „ third molars.

One quarter of the mouth may be represented diagrammatically thus :

Teeth ..	I.	I.	C.	M.	M.	Teeth .	I. I. C. B. B. M. M. M.
Month of ⎱ eruption. ⎰	6	9	18	12	24	Year of ⎱ eruption. ⎰	7 8 11 9 10 6 12 24

The normal order of eruption of teeth may be represented thus : MILK teeth, 6, 9, 18, 12, 24 MONTHS ; and PERMANENT teeth, 7, 8, (11), 9, 10 ; 6, 12, 24 YEARS. These details are worth remembering, because defective or deficient teeth are nowadays an extremely frequent cause of faulty digestion.

Septic teeth, dental caries and pyorrhœa alveolaris are amongst the commonest causes, if not the commonest, of the dyspepsia of modern times, and it is an ominous feature that a very large proportion of the candidates for the Army and Navy Services are rejected because of bad teeth. The causes of dental caries are somewhat obscure, but amongst the recognised conditions are : (1) in the infant, faulty feeding, especially the absence of vitamins in the diet. When a child is properly fed with a correct proportion of vitamins in his diet, the first and second dentitions are healthy. (2) Neglect of proper cleansing and care of the teeth is a common cause of dental caries. Research rather points to the fact that the prevalence of sweet and farinaceous pulpy food is a prime factor in the causation of dental caries. (3) Dental caries frequently follows a lack of care of the teeth during illness, especially in cases of prolonged pyrexia.

The teeth are altered in shape after stomatitis in early life, which may be due to mercury, etc. (*vide* § 170). In these circumstances the teeth present transverse and vertical ridges, with or without alteration of shape. " Hutchinson's teeth " show alterations in the shape of the permanent teeth, due to hereditary syphilis, and present a valuable means for the identification of this disorder, as they are of very frequent occurrence in that disease, and bear lifelong testimony. They are set apart and are

both pegged and notched, the transverse measurement is smaller at the
• free edge than the part near the gum, and on the edge of each tooth
there are notches. The onset of acromegaly may be detected by alteration
of the " bite " of the teeth owing to the pushing forward of the jaw.

§ 167. **Toothache** (odontalgia) is caused most frequently by decay
(caries) of the teeth ; but there are OTHER CAUSES, the chief of which are :

1. Morbid conditions of the *tooth-pulp*, including acute and chronic inflammation,
pressure from confined matter in the pulp cavity, and deposit of secondary dentine
in its substance. 2. Exposure of sensitive dentine, with or without caries. 3. Morbid
conditions of the peridontal membrane, odontoma, exostosis, acute and chronic
alveolar abscess, and impacted teeth. 4. Morbid conditions of the *periosteum of the
jaw—e.g.*, traumatic, rheumatic, syphilitic ; or residual infection after removal of
diseased teeth. 5. Irritation of the *dental nerves* without visible local lesions—*e.g.*,
malposition and retarded eruption of wisdom teeth, pressure due to insufficient space
for the teeth. 6. *Inflammations and ulcerations of the mucous membrane.* 7. In an
acutely *inflamed antrum* the premolar and molar teeth will ache.

This is merely an approximate classification. The character and degree of the
pain is greatly modified by the condition of the patient. The pain is generally more
or less intermittent. It is often absent at periods of full vigour—*e.g.*, after breakfast
or dinner. Pain due to irritation, or to chronic local inflammation of the pulp, is
of a neuralgic character, and the patient is often unable to point out the affected
tooth or teeth. The suffering induced by acute inflammation of the pulp is excessive,
particularly if there be no exit. It ceases more or less abruptly from the consequent
death of the pulp. The recumbent posture or active exercises aggravate the pain by
increasing the vascular supply.

The *Treatment* belongs to the dental surgeon, but a good deal of temporary relief
may sometimes be obtained by constantly rinsing the mouth with hot carbolic lotion
(1 in 100). A formula for drops to apply to a hollow tooth is given in Formula 23
at the end of this book.

Although the term *Epulis* should be restricted to new growths of a malignant
character springing from the alveolar portion of the jaws, it is often employed to
indicate any proliferation of tissue in that situation. True epulis is a fibro-sarcoma
growing from the periosteum. It spreads along the gum, and should be excised early.
A myeloid growth from the cancellous tissue of the jaw may simulate epulis ; so
may an epithelioma. In leukæmia the teeth may be almost hidden by profuse pro-
liferation of the gum.

The Gums.—The pallor of anæmia, the stippled blue line of lead-
poisoning, the red and ulcerated condition in stomatitis, the sponginess
in mercurialism and scurvy are all useful local indications of some general
condition. A swelling of the gums with greenish discharge suggests
Actinomycosis. *Bleeding from the gums* is apt to occur in scurvy, purpura,
the hæmorrhagic diathesis, and even in apparent health, when the teeth
are covered with tartar and the gums recede. In some people the gums
very readily bleed, and sucking them may produce bleeding, which enables
malingerers and hysterical persons to simulate diseases of the lungs or
stomach. It is detected by appearing only in small or moderate quantity,
and by its intimate mixture with saliva. Bleeding on brushing the teeth
may be the earliest sign of Pyorrhœa Alveolaris—the importance of which
has only recently been recognised.

§ 168. **Oral Sepsis.**—Under this heading come certain affections of the

teeth and gums. Of the following conditions the first two are very common: (1) Apical abscesses, (2) pyorrhœa alveolaris, (3) suppurating impacted teeth. This is especially met with in connection with the lower wisdom teeth ; X-ray reveals the condition when not suspected from clinical signs. (4) Suppurating cysts. Most serious trouble is caused by *apical abscesses*, which occur at the roots of the teeth. This condition is frequently entirely unsuspected ; the patient may feel no pain and no tenderness is elicited on tapping the affected teeth. In cases of chronic ill-health without discoverable cause, a radiogram may reveal the existence of one or more apical abscesses. They are the focus of infection in many cases of rheumatoid arthritis, and sometimes in iritis, recurrent gastric or duodenal ulcer, anæmia and menorrhagia.[1] Even neurasthenia has sometimes been traced to this cause.

§ 169. **Pyorrhœa Alveolaris** is an ulcerating condition of the gums around the sockets of the teeth or stumps. When tartar is allowed to collect upon the teeth, it gradually pushes the gum back ; by degrees a pocket or fossa is formed around the neck of each tooth, with considerable sero-purulent and often blood-stained discharge from the pockets thus formed. This imparts an offensive odour to the breath, and, being continually swallowed, causes a chronic toxæmic condition and infects the stomach and intestine. Pyorrhœa may be present when to the eye the gum looks natural ; on pressure pus oozes out by the teeth. Dyspepsia, even apart from difficulties of mastication, invariably ensues sooner or later. Even before the dyspepsia becomes established the patient is listless, languid, and unfit for work, and complains of functional nerve symptoms. Other cases do not suffer because they can resist the invasion, but at any rate their threshold of resistance may become lowered. A large proportion of functional neuroses are due to oral sepsis. Among the symptoms due to this cause are headache, neuralgia, pains or tingling in the limbs and prostration, attacks of flushing or giddiness ; a feeling of heaviness, and swelling of the limbs which is sometimes attended by œdema of the ankles, wrists, and other parts, which differs from ordinary anasarca in requiring longer pressure to produce the pit. Great depression, even melancholia, may result. Rheumatoid arthritis, iritis, colitis, purpura, and fatal anæmia may also occur.

Treatment.—If the cause be not removed, no treatment is of much use. Autogenous vaccines have been employed, and ionisation has given good results. Emetine is good where the infecting organism is an amœba. Local treatment in every case should be carried out by the dentist. The physician attends to the general and remote effects.

Vincent's **Gingivitis** is an infection of the gums, localised usually to a few teeth, due to the spirillum of Vincent's Angina (§ 129).

§ 170. **Stomatitis** is a generalised inflammation of the mouth, evidenced by redness, swelling, tenderness, and pain of the mucous membrane, swelling and protrusion of

[1] I have seen two cases of severe metrorrhagia due to oral sepsis.

the lips in severe cases, offensive odour of the breath, and usually, but not always, excess of saliva. This, the simplest form of stomatitis, such as occurs in dentition or the application of caustics, is known as (*a*) *Catarrhal* or *Erythematous Stomatitis*. (*b*) *Aphthous Stomatitis*, also known as *Vesicular* or *Herpetic Stomatitis*, occurs in badly-fed children, and it presents, in addition to the above features, small grey patches, with a red base and sharply-defined circular margin, resembling vesicles, which may be very painful to the touch. (*c*) *Ulcerative Stomatitis* occurs in a mild, and also in a severe form. In this we find, in addition to the features belonging to variety (*a*) irregular ulcers, especially on the gums which recede from the teeth, so that the teeth become loosened. In the severe form there is a great fœtor of the breath, considerable enlargement of the glands, submaxillary and cervical, and constitutional disturbance ; and the teeth may drop out of the ulcerating gums. The ulcers often have a yellowish or grey coating, resembling a membrane, and it is therefore sometimes called " pseudo-membranous stomatitis," or " phagedenic gingivitis." (*d*) *Gangrenous Stomatitis* (Cancrum Oris, Phagedena Oris, Noma Oris) is a gangrenous inflammation starting at one spot, usually on the cheek or on the lips. At first there is acute pain, but as this passes off a black spot forms (usually both internally and externally), which spreads and leads to perforation of the cheek. The inflammation may spread to the gums, and the teeth become loosened. This is a severe disease, attended by considerable prostration, and, at first, a subnormal temperature. It is apt to follow measles or other exhausting illnesses in weakly children exposed to bad hygienic conditions. *Pyorrhœa Alveolaris* has been separately described above (§ 169).

Etiology of Stomatitis.—(1) Certain *local conditions*, of which the commonest are dentition, tartar, and a want of cleanliness, the local irritation of a jagged tooth, excessive smoking, dirty feeding-teats in children, the application of hot fluids and caustics, new growths (simple or malignant), and gummata. In most of these cases the stomatitis takes the form of (*a*) or (*b*) above. Mouth-breathing and chronic gastric catarrh are said also to give rise to stomatitis occasionally, and necrosis of the jaw may lead to an ulcerative stomatitis.

(2) Certain *drugs and chemical substances* are apt to cause stomatitis. Chief amongst these is mercury, which gives rise to a very characteristic ulcerative stomatitis, with spongy gums and great fœtor of the breath. Arsenic and iodides may produce catarrhal stomatitis. Phosphorus produces ulcerative stomatitis, with necrosis of the jaw. The blue line of lead may be attended by a certain amount of catarrhal stomatitis.

(3) Chief among the *constitutional conditions* which cause stomatitis is (i.) the lowered vitality met with in phthisis and other wasting disorders, or in badly-fed children, in whom the stomatitis may be aphthous or ulcerative. Thrush often accompanies catarrhal stomatitis in these circumstances. (ii.) Syphilis is accompanied by a special variety of the catarrhal form, and is attended by whitish, semi-transparent patches on the tongue and mucous membrane, resembling " snail-tracks." Later on ulcerations may occur (§ 129, Throat). It may also take the form of flattened white papules. (iii.) Measles and other acute specific fevers are apt to be followed by cancrum oris in children exposed to bad hygienic conditions. Diphtheria is attended by both stomatitis and rhinitis when the membrane affects the mouth and nose. (iv.) Scurvy and purpura are attended by swollen and spongy gums and ulcerative stomatitis. The acute blood diseases (acute lymphæmia and myelæmia) are accompanied by an extreme degree of stomatitis, due to the presence of small pin-head to sago-grain-sized lymphoid nodules on the gums which readily ulcerate. Not infrequently such cases are treated without any suspicion of the true nature of the disease, although the nodules in question are very characteristic. (v.) Gastro-intestinal derangement, as in dyspepsia and fevers, leads not infrequently to catarrhal and sometimes aphthous stomatitis. (vi.) A lowered state of health, with insanitary environment, gives rise to epidemics of ulcerative stomatitis, sometimes taking a fatal form, in jails, hospitals, and camps. Occasionally this condition is met with in individuals in private life. (vii.) *Foot-and-Mouth Disease* (Syn. : epidemic stomatitis ;

aphthous fever) is an acute infectious disease attacking pigs, sheep, cattle, and other domestic animals. Epidemics have been reported in which the disease was transmitted to man, with symptoms of fever, gastro-intestinal derangement and vesicles on the lips, mouth, and pharynx, and sometimes near the nails of fingers and toes. Death has occurred, but recovery is the rule. (viii.) Sprue, Pellagra and Pernicious Anæmia commonly show stomatitis. (ix.) With *Espundia*, in S. America, oropharyngeal ulceration follows the primary skin lesion, which is due to a type of Leishmann-Donovan body, transmitted by a bug. Tartar emetic is specific.

(4) Certain *skin lesions* may invade the mucous membrane of the mouth, such as the rashes of small-pox, chicken-pox, measles, and herpes iris. In measles certain spots, first described by Koplik, appear on the inner sides of the cheeks, opposite the bicuspid or molar teeth, before the skin eruption occurs. They most often take the form of a greyish-white stippling on a slightly raised purplish base, and afford considerable aid in the early diagnosis of the disease. Lichen ruber planus may affect the mucous membrane of the mouth and tongue, and it may be present there long before it appears on the skin. In this situation it has a whitish appearance much resembling secondary syphilis, for which it has sometimes been mistaken. Lupus may affect the palate.

Prognosis of Stomatitis.—As a rule, stomatitis is not a serious disease, except that form known as phagedenic stomatitis, in which the mortality is 80 per cent. Catarrhal, aphthous, and ulcerative stomatitis generally end in recovery in a week or two. Those cases due to constitutional conditions are, as a rule, far more serious and obstinate than those due to local or removable conditions. The stomatitis of mercury may be extremely severe, but is, happily, only rarely seen nowadays. When aphthous stomatitis occurs in adults, accompanying a lingering disease, it is very obstinate, and is, in itself, a very grave omen. The prognosis is grave in the epidemic form, which is probably of microbic origin. The complications of the phagedenic form are diarrhœa, broncho-pneumonia, and gangrene in other parts of the body, especially the organs of generation (noma pudendi).

Treatment.—In all varieties (1) to remove the cause, (2) alleviate the local inflammation, and (3) attend to the general health. The teeth should be scaled and any septic stumps removed. Carious cavities and ulcers on the gums should be thoroughly swabbed over with 1 in 40 carbolic. It is important to cleanse the pockets at the sides of the teeth which are met with in pyorrhœa alveolaris. After every meal the mouth should be cleaned of débris with a soft brush, by rinsing repeatedly with warm water, then with an antiseptic solution. One of the best is hydrogen peroxide (2½ to 10 vol.); others are 1 in 200 carbolic in normal saline, 1 in 30 boracic, and sodium bicarbonate 1 in 20. When swelling is pronounced and prevents free access of these remedies, a 2 per cent. solution of chromic acid swabbed over is highly recommended. If the mouth is very dry, glycerine of borax is useful. A " bad taste in the mouth " may sometimes be overcome by taking ℔ i. of pure carbolic in ℥ſ. (0·06–32) water twice daily. Tablets containing formalin may be sucked at frequent intervals, and are of especial use when dealing with children.

Aphthous and Ulcerative Stomatitis are best treated by touching the sore places with solid nitrate of silver or sulphate of copper. For the pain, a solution of cocaine (½ to 3 per cent.) may be used. In the ulcerative form chlorate of potash is especially useful. In the *Gangrenous* form (cancrum oris) prompt measures are necessary to avert a fatal issue. The affected area should be as freely excised as possible, and any suspicious tissue left or subsequently appearing may be burnt with the actual cautery. Plastic operations may be necessary later, but attempts to save tissue at the time are always fatal. Free use of stimulants and nourishment is called for.

The Tongue

The alterations to which the tongue is liable will be referred to under six headings : (*a*) Furring of its Surface ; (*b*) Ulceration ; (*c*) White

Patches ; (d) Acute Swelling; (e) Chronic Swelling (Hypertrophy), and
Atrophy; (f) Warts, Fissures, and Cicatrices. A mother sometimes
speaks of her child being " tongue-tied " when the frenum is too short. In
some cases this is really so, or the structure may be attached to the tongue
too far forward, but it exists much less frequently than parents suppose.

§ 171. Furring of the Tongue.—The appearance of the dorsum of the
tongue used to be looked upon by older authors as an indication of the
state of the stomach, and with certain reservations it is still regarded as
some aid in the investigation of that organ (§ 219), though it is a better
guide in the prognosis of fevers and other grave constitutional disorders.
Five varieties of tongue have been described by authors : (1) The pale,
large, flabby tongue, with broad tip and indented edges, and a uniform
thin white coating, is the commonest abnormality. It is met with after
alcoholic excesses, in atonic dyspepsia, in anæmia, and in gouty persons.
(2) A red tongue, with sharp red tip and edges, in which the hyperæmic
papillæ contrast strongly with the slight white coating in the centre, is
found in subacute gastritis and hyperchlorhydria. (3) The coated tongue,
with a uniform white layer over the surface, is found in acute gastritis,
feverish conditions, anæmia, constipation and nervous depression. Two
forms have been described : (i.) The strawberry tongue, having a slight
white coating through which the fungiform papillæ protrude at the tip
and edges, is very typical of scarlatina and other highly febrile states.
(ii.) The plastered tongue, where the coating is considerably thicker.
The amount of coating on a tongue varies directly with (a) the amount
of dryness of the mouth—that is to say, the deficiency of salivary secretion
(e.g., in fevers and profuse perspiration) ; and (b) with the immobility of
the tongue, owing to eating food that does not require mastication. The
plastered tongue may pass on to—(4) the furred tongue. The coated
papillæ stand out separately, giving a shaggy appearance. It is met
with in states of marked prostration—e.g., coma, abdominal cancer,
advanced phthisis, profound anæmia, and other asthenic states. The
prognosis is grave when the tongue becomes encrusted and its dryness
increases. From any cause it may become dry, brown, and crusted, and
then pass on to—(5) the denuded red tongue, which generally follows the
preceding as the crust falls off. This tongue is red, shiny, smooth, and
often cracked. It is found in advanced states of the preceding conditions,
in diabetes, and other severe chronic ailments. The appearance of this
·tongue in a disease is of very grave prognosis. Aphthous stomatitis may
supervene. (6) A pale tongue with marked atrophy of the mucous
membrane and tender minute ulcers is often seen in association with
Addisonian (pernicious) anæmia.

Apart from disease, there is no doubt that there are wide individual
peculiarities in the character of the tongue. On this account some [1] go so
far as to say that the tongue is of little importance as a clinical indication.
Undoubtedly we should make sure in any given case that the tongue

[1] E.g., Sir Jonathan Hutchinson, Med. Press and Cir., July, 1883,

condition before us is not due to these personal peculiarities, to smoking in excess, or to previous disease.

As regards *Treatment*, it is an old saying that a red tongue requires alkalies, and a white tongue acids. The former alone is true to some extent. With the exception of diabetes, a dry tongue indicates no appetite, and deficient gastric secretion ; therefore the patient should be fed on fluids, animal soups, and other things requiring no great digestive power ; (4) and (5) call for alcohol and other stimulants. In the prognosis of typhoid the tongue is a valuable indication ; in severe cases it is shiny, dark brown, dry and hard.

That rare condition, *black* or " *hairy* " tongue, must not be mistaken for a furred tongue. It is due to elongation of the papillæ at the back of the tongue ; they resemble dark hairs. The cause is unknown. The condition is best left alone, as it usually disappears spontaneously.

§ 172. **Ulcers of the Tongue** may be Simple, Syphilitic, Malignant, or Tuberculous.

I. SIMPLE ULCERS of the tongue are known by their superficial character, by the presence of some local cause, such as a jagged tooth or other local irritation (see also Ulcerative Stomatitis). The *frenum* is apt to be ulcerated in whooping-cough. This is due to friction against the lower teeth, and is a useful aid in diagnosis.

II. SYPHILITIC ULCERS are of two kinds : (*a*) superficial, (*b*) deep.

(*a*) *Superficial Syphilitic Ulcers* of the tongue are met with usually at the side, or in the form of fissures on the dorsum (*cp.* § 132) or superficial circular " punched-out " ulcers.

(*b*) *Deep Syphilitic Ulcers* are preceded by the formation of a roundish nodule (a gumma) which ulcerates. They are recognised by (i.) their site, which is usually on the centre of the dorsum ; (ii.) their raised, ragged, and sometimes undermined edges ; (iii.) the yellow slough which covers the base ; and (iv.) the fact that they leave deep stellate scars. Syphilitic ulcers are usually multiple ; difficulty in diagnosis arises in the case of a single ulcer as to whether it be syphilitic or cancerous. Syphilitic ulceration is differentiated by (1) the relative absence of surrounding induration, and consequently less interference with the movements of the tongue ; (2) the site of the ulcer on the dorsum ; (3) less glandular enlargement, and the glands have a shotty feel ; (4) the age of the patient, malignant ulcers rarely occurring before forty ; (5) little or no pain is felt ; and (6) there is a history of syphilis, and the disease *heals under iodide of potassium.*

III. MALIGNANT ULCER of the tongue is known by (i.) its site, which is chiefly on the side of the tongue ; (ii.) its hard, raised, everted edges, and its uneven warty base, with foul discharge and tendency to hæmorrhage ; (iii.) the induration around, and the early involvement of the glands ; and (iv.) the early impairment of the movements of the tongue, with great pain. These are the characters in an advanced case when

diagnosis from syphilis is relatively easy. In an early stage it may be very difficult. In that stage a cancerous ulcer has flat sloping edges and scanty secretion, *its progress is very slow*, and it does not yield to iodides. Before a suspicious ulcer has existed for any length of time, a small piece should be excised for microscopic examination.

IV. TUBERCULOUS ULCERS are not common. They are superficial, with a yellowish discharge, usually near the tip, and there is generally a history of tubercle in the lung or throat. The tubercle bacillus may be found in the scrapings.

Prognosis.—Simple ulcers are easily dealt with, but other ulcers of the tongue are dangerous chiefly from their liability to hæmorrhage and because of the important structures around. The diagnosis of syphilitic from malignant lesions is as important as it is difficult, for however advanced the former may be, they yield to appropriate remedies, but the latter are necessarily fatal unless removed early. The deep ulcers often seen in advanced syphilitic glossitis are dangerous, as the deeper parts may be affected by malignant change.

The Treatment consists of the usual surgical measures or diathermy. In syphilitic ulcers the usual anti-syphilitic remedies must be given. Potassium iodide should be taken in large doses—gr. x–xxx (0·6–1·8) ; sufficient is rarely given.

§ 173. White Patches are not infrequently met with on the tongue, and may result from : I. Thrush ; II. Leucoplakia ; III. Geographical tongue; IV. Aphthous Stomatitis (§ 170); V. Syphilitic Patches (§ 132). The two last are described elsewhere. The stellate cicatrices so characteristic of syphilitic lesions must not be confused with any of these.

I. In THRUSH (parasitic stomatitis) there are white membranous patches, like milk curd, sometimes with an areola round them. They are distinguished from other similar affections by (i.) leaving a bright, bleeding surface when they are scrapped off, and (ii.) by the detection of the fungus *Oidium albicans* (*Saccharomyces albicans*, Fig. 58) on microscopical examination. It usually starts on the tongue, but may invade the lips and the whole of the interior of the mouth. The disease occurs chiefly in infancy, also in the later stages of exhausting diseases in adults. In the adult it only occurs at the end of wasting disorders, and not infrequently forms one of the indications of approaching death.

FIG. 58.– OIDIUM ALBICANS OR THRUSH FUNGUS.

In infancy it generally arises in hand-fed children under bad hygienic conditions, and is often attended by diarrhœa. It is contagious from child to child. In children it has no very great significance, and readily yields to glycerine and borax, or weak carbolic lotion (1 in 500). The diet and method of feeding should always receive attention in such cases. In such children it sometimes happens that excoriations are noticed around the anus, and the mother thinks the "thrush has gone through the child " ; but these are more frequently due to congenital syphilis or eczema intertrigo.

II. LEUKOPLAKIA LINGUÆ (synonym, Ichthyosis Linguæ) is a term applied to flat,

whitish, horny-looking, silvery patches on the tongue, due to a heaping up and condensation of the epthelium. The disease generally involves a considerable area of the tongue. In a later stage the tongue becomes red and glazed. The patches themselves are often cracked, and form a pavement-like surface, which has the appearance of ichthyosis of the skin. They give rise to a great deal of discomfort and tenderness. It is most frequently met with in tertiary syphilis, but is sometimes attributed to excessive smoking, jagged teeth, drinking, and dyspepsia. I cannot say that I have met with a case which could not be attributed to syphilis. The *Treatment* is, as a rule, very unsatisfactory, unless the disease be met with in the early stages. A mouth-wash, consisting of bicarbonate of soda, 20 grains to the ounce (1 in 24), or a saturated solution of chlorate of potash, sometimes relieves the symptoms. Superficial cauterisation is the best method of treatment; failing that, the local application of chromic acid (1 or 2 per cent.), painted on daily does good. It should be accompanied by antisyphilitic remedies, though they do not have a very marked effect. Ionisation with zinc chloride or salicylate of soda is also recommended. Alcohol, smoking, and other irritants must be avoided. The tongue should be carefully watched lest malignant disease supervene.

III. GEOGRAPHICAL or "Mapped" tongue is a condition in which the normal desquamation of the tongue takes place irregularly, with the formation of more or less circular patches surrounded by margins of slightly proliferating whitish-grey epithelium. The cause is unknown. It may disappear spontaneously.

§ 174. Acute Swelling of the Tongue—*i.e.*, swelling of the tongue coming on rapidly —may be due to either (*a*) *Acute Glossitis* or (*b*) *Acute Œdema*. In both of these the tongue rapidly enlarges, and may even protrude beyond the teeth. A great deal of pain is present, and there is a difficulty of swallowing and speaking.

(*a*) ACUTE GLOSSITIS may be due to various local causes—*e.g.*, the sting of an insect, streptococcal infection from the teeth, biting or wound of the tongue, acute ulcers—or it may be due to constitutional conditions—*e.g.*, mercurial salivation, and, according to some, acute specific diseases, such as erysipelas. The onset of acute glossitis is rapid, though not so rapid as in acute œdema; the swelling extends to the neck, and the glands becomes involved. *Treatment* must be prompt, to avert a fatal issue. Ice at first, then hot fomentations. Serum must be given early, then vaccines. Free incisions may be necessary.

(*b*) ACUTE ŒDEMA OF THE TONGUE is a serious disorder, because of its liability to involve the glottis. It may accompany urticaria, angio-neurotic œdema, or it may be, like the Angina Ludovici (§ 134), of an erysipeloid nature. The œdema comes on suddenly, and in the course of a few hours the tongue may protrude from the mouth. The swelling rapidly extends to the throat, nose, and down the œsophagus and trachea. It is attended by an inability to speak, to swallow, and sometimes even to breathe. Its *Causation* is obscure, but it is said to be usually of an urticarial nature, and to occur in those who have had urticarial attacks. This condition is *Diagnosed* from simple acute glossitis by (i.) its rapid advent in the course of an hour or two ; (ii.) the rapid extension to the throat and other parts ; (iii.) the presence sometimes of an urticarial rash, or a history of sensitiveness to some article of food (§ 485). Without the last feature the diagnosis is difficult.

Prognosis and Treatment.—The disease comes on rapidly, and runs a very rapid course, subsiding in the course of twenty-four hours, unless the patient die in the meantime. It is apt to cause suffocation. Prompt measures are necessary. A strong purge should be given at once (croton oil, 1 minim, if it can be swallowed) or a turpentine enema. Cocaine (5 or 10 per cent.) or adrenalin should be kept constantly painted on the tongue. Scarification is often required, and the practitioner should be prepared to perform tracheotomy if necessary.

§ 175. Chronic Swelling, Hypertrophy and Atrophy of the Tongue.

I. *Chronic Glossitis* is a chronic inflammation of the tongue, in which either the surface or the substance is mainly involved. The *surface* is covered with irregular,

red, raw, tender patches and cracks ; later, it becomes smooth and glazed. If the *substance* be affected, the organ is enlarged, indented by the teeth, and in course of time it becomes indurated. It is more frequently due to some local irritation, such as a jagged tooth or an ulcer, in which case the enlargement is generally limited to one part of the tongue. Glossitis arises from infection by streptococci, saccharomyces or bacillus Friedlandler, from alcoholism, syphilis, chronic dyspepsia, or excessive smoking, and other causes of stomatitis (*q.v.*). *Treatment* is directed to the removal of the cause, and the employment of soothing applications after meals, such as olive oil and tr. hammamelis. *Tumours* of the tongue are rare ; for diagnosis and treatment of these a surgical work must be consulted. Overgrowth of the lymphadenoid tissue at the base of the tongue (the " lingual tonsil ") is found in local septic conditions and acute blood diseases. Rarely, thyroid tissue remains at the base of the tongue as a developmental defect.

II. *Macroglossia* is due generally to a congenital overgrowth of the connective tissue, accompanied by a dilatation of the lymphatics of the tongue. Its causes are obscure. It is found in mongolism and with acquired syphilitic lesions. Persistent application of mild caustics or the galvanic cautery to the tongue is the only remedy.

III. *Atrophy of the Tongue* (microglossia) may arise from nerve lesions. It may occur in bulbar paralysis, and is then usually bilateral. In unilateral cases the lesion is either situated in the nucleus or trunk of the twelfth nerve of one side (*vide* Chapter XIX, § 662).

§ 176. **Warts, Fissures, and Cicatrices.** Warts are simple or syphilitic. *Simple* warts are distinguished by the fact that they are soft ; they are raised, and often pedunculated, and there is but little secretion. The glands are not shotty to the touch. *Syphilitic* warts are hard, with infiltration ; they are never pedunculated, secretion is present, and the glands in the neck and elsewhere are shotty. FISSURES are also divided into simple and syphilitic. The *simple* can generally be accounted for by some such cause as the irritation of a ragged tooth, and are never infiltrated. On pinching *syphilitic* fissures between the fingers, infiltration is found to be present. CICATRICES.—Simple ulceration rarely leaves a scar, but if so, it is never hard. Hard, stellate scars are invariably indicative of syphilis.

The Gullet

§ 177. **Symptomatology.**—Diseases of the œsophagus have practically one symptom which is common to all—namely, *dysphagia—i.e.*, a difficulty in swallowing. There are certain features about this symptom which it is important to investigate :

First, does the difficulty apply to both liquids and solids ? This gives us an idea of the *degree* of the obstruction. *Secondly*, does the food return ? and if so, after what interval ? This is sometimes a guide to the *seat* of the obstruction. Obstruction within the *œsophagus* has to be distinguished from obstruction at the pyloric end of the *stomach* (i.) by the easy way in which food regurgitates as compared with the vomiting which accompanies pyloric stricture ; and (ii.) by the absence of acidity in the material returned. *Thirdly*, is there any pain ? Its situation aids diagnosis of the position of the growth. Is it present only after the ingestion of food ? Constant pain is a feature of malignant disease. *Fourthly*, what is the duration of the dysphagia ? Has it been persistent, and become progressively and steadily worse ? The last named is the leading feature of organic, as distinguished from functional, dysphagia, which is

frequently intermittent, and by no means progressive. *Fifthly*, is there any regurgitation through the nose ? This feature implies paralytic dysphagia, with paralysis of the soft palate. *Sixthly*, is there any emaciation, or are there any symptoms referable to other organs ? Marked emaciation coming on early in a patient beyond middle life is characteristic of carcinoma.

§ 178. **Physical Examination.**—(*a*) A careful *inspection* of the throat should be made, because the dysphagia may arise from tonsillitis or other pharyngeal conditions. The paralysis of the palate which succeeds diphtheria may thus be detected. Any swelling should be carefully examined, such as retro-pharyngeal abscess or tumour of a foreign body in this situation. I have known the bristle of a toothbrush entangled in the pharynx give rise to very serious difficulty in swallowing.

(*b*) In cases of dysphagia of any duration a skilled X-ray examination with a bismuth meal is advisable, but where this cannot be obtained a bougie or a soft stomach tube should be passed. The solid bougie is preferable, but if carcinoma is suspected, great care must be exercised. The chest should always first be examined for aneurysm,[1] and if this cannot certainly be excluded, the bougie should be avoided. The bougie must first be dipped in hot water in order to make it more flexible, and glycerine if necessary for lubrication. There is not much fear of it entering the larynx, provided the tube be passed to one or other side, and instruction given to the patient to put his head horizontally forwards and swallow during the operation. As the entrance to the stomach—from the teeth to the cardiac orifice—is a distance approximately of 16 inches, it is a good plan to tie a thread round the bougie 16 inches from its point ; then one can tell when it has reached the stomach. The œsophagus starts at the cricoid cartilage, opposite the sixth cervical vertebræ, and ends opposite a point between the ninth and tenth dorsal vertebræ, a distance of 10 inches. The presence of acute *pain* during the passage of the instrument indicates ulceration, either simple or malignant. The presence of *blood*, and perhaps cancer cells adhering to the end of the tube, should be looked for as having the same significance as the foregoing. The presence of *dilatation* may be suspected when the end of the tube is not gripped, but is loose and easily movable. Occasionally a diverticulum or saccule of the œsophagus is formed, which by its pressure on the gullet above or below it causes obstruction. In such cases a bougie, which could not be passed before, may be passed after vomiting has occurred.

(*c*) *Auscultation* affords a valuable means of detecting both the presence and position of an œsophageal stricture. Place the chest end of a binaural stethoscope over the interval between the xiphoid cartilage and the left costal arch. Two gurgling sounds can be heard in this situation if the

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[1] Dr. J. S. Bristowe, with characteristic candour, narrates a case shewing the consequences which arose from a neglect of this procedure in his " Clinical Lectures and Essays," p. 43. The case was really one of dilatation of the œsophagus, which remained undiscovered until after death.

patient swallows *one* gulp of fluid ; the first is when it passes from pharynx to œsophagus, the second is when it passes from œsophagus to stomach. The normal interval between these two is *six seconds*, but if there be any obstruction in the gullet this interval becomes increased. If the first sound cannot be distinctly heard, the moment of its occurrence can be judged by looking at the throat. Again, by placing the stethoscope on the left side of the neck in a healthy person a gurgling sound will be heard during the act of swallowing. This normal sound may be *traced round and down the back* on the left side of the vertebral spines as low as the tenth dorsal vertebræ. But if a stricture be present it will be delayed *or absent below the seat of stricture.*

(*d*) X-ray examination with a bismuth meal is an important method for detecting the presence of stricture or diverticulum.

(*e*) The œsophagoscope may be used by skilled hands.

§ 179. **Causes of Dysphagia.**—*" When a patient complains of difficulty in swallowing, or that the food returns to his mouth, the practitioner should first think of thoracic aneurysm, secondly of cancer, and thirdly of some kind of ulceration."* [1]   The COMMONER CAUSES are—

      I. A tumour pressing upon the gullet from the outside.
      II. Cancer of the gullet.
      III. Simple or non-malignant stricture.
      IV. Spasm.
      V. Foreign bodies, acute œsophagitis, and simple ulcer.

LESS FREQUENT CAUSES are—

      VI. Paralysis of the gullet.
      VII. Dilatation or diverticulum of the gullet.

§ 180. **A Tumour** pressing upon the gullet from without is perhaps the commonest cause of dysphagia, although malignant or simple stricture and muscular spasm are regarded by many as of equal frequency. Any intrathoracic tumour may, by its pressure, narrow the lumen of the gullet, and undoubtedly the commonest of these is aneurysm of the aorta. Other tumours are cancer of a neighbouring viscus, retropharyngeal abscess or tumour, enlargement of the bronchial glands, lympho-sarcoma or other mediastinal tumour, goitre, pericardial effusion, and diverticula of the gullet filled with food (§ 186). The features common to all such tumours are the slowly progressive character of the dysphagia, the symptoms of pressure on other viscera, and sometimes, although usually not until late in the case, the physical signs of the tumour in question. For the rest, the differential features vary according to the nature and position of the tumour. In *aortic aneurysm* the amount of dysphagia is rarely very great at any time, although it is slowly progressive. Rest in bed will generally ameliorate the dysphagia. Difficulty of swallowing is only

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[1] Bryant, quoted by Fagge and Pye-Smith, " Prin. and Pract. of Med.," vol. ii., p. 316, second edition.

one of the pressure symptoms in this disease, and others should be looked for—*e.g.*, dyspnœa, abductor paralysis of the left vocal cord, and inequality of the pupils, and the radial pulses. The physical signs of aneurysm are commonly wanting in such cases on account of its deep-seated position.

§ 181. **Malignant Disease** of the œsophagus is due in the large majority of cases to an epitheliomatous growth in the wall, usually primary, which goes on to ulceration, and forms a stricture from 1 to 4 inches long. Rarely the growth is sarcomatous. The diagnostic features of epithelioma of the œsophagus are : (i.) The patient is past middle life. It is said to be more common in males. (ii.) The dysphagia becomes steadily and progressively worse ; in rare cases it may be intermittent. At first a difficulty exists only with solids, but later on fluids also are returned. The duration of the whole illness rarely exceeds twelve to eighteen months. (ii.) Emaciation and other evidences of cachexia occur quite early in the illness, owing to deficient nourishment. (iv.) There may be evidences of cancerous deposit elsewhere, especially within the abdominal cavity ; or there may be enlarged glands, especially above the left clavicle. (v.) Pain and hæmorrhage, those frequent accompaniments of all malignant growths, are usually present and the pain is persistent and independent of, although aggravated by, food. (vi.) The passage of a bougie is attended by considerable difficulty. The favourite sites of malignant stricture are opposite the cricoid cartilage, 6 inches from the teeth ; opposite the bifurcation of the trachea, 9 inches ; and at the cardiac orifice, 16 inches from the teeth.

Fibroma and *Myoma*, and other benign growths in the œsophagus, sessile, or in the form of polypi, are very rare. They may simulate simple or malignant stricture, and there may be hæmorrhage ; but the absence of any cachexia and the long duration without any increase of symptoms are the only means of suspecting the condition.

§ 182. **Simple or Non-Malignant Stricture** of the œsophagus is most frequently caused either by the narrowing due to a syphilitic infiltration or the contraction which it subsequently leaves. It may also arise from the cicatrisation which follows a simple ulcer of the gullet or stomach ; or, thirdly, as the result of swallowing a corrosive liquid. Dilatation may take place above the stricture. The differential features of this condition are : (i.) The dysphagia comes on gradually, and, having reached a certain degree, is apt to remain stationary ; the patient may be unable to swallow solids, but lives for many years on liquid food. (ii.) The passage of bougies gradually increased in size is possible, and this procedure gives some relief. (iii.) The patient may be young, or he may be of any age ; the cachexia of cancer is wanting ; and pain is not a prominent feature in the case. (iv.) The gullet is apt to dilate above the stricture, and the food returns after an interval, which becomes progressively longer as the dilatation becomes greater. (v.) There is nearly always a history of one of the three causes above mentioned.'

§ 183. **Spasm of the Pharynx or Œsophagus** is, in the author's experience, one of the commonest causes of dysphagia. It is not infrequently

associated with hysteria and other functional neuroses.   Its differential features are fairly characteristic : (i.) The dysphagia is never progressive.   It may come on somewhat suddenly, dating perhaps from an emotional shock or trouble, and it is very often intermittent, the patient being well enough in the intervals.   Sometimes solids can be taken, while fluids are regurgitated, or *vice versa*.   (ii.) It is unaccompanied by emaciation or cachexia ; indeed, the patient sometimes appears to be in perfect health, a feature in which it differs from all other causes of dysphagia.   There is usually little or no pain, and never any bleeding.   (iii.) The dysphagia may last intermittently for a considerable time.   I have known cases persist in varying degree for seven, twelve, and sixteen months.   (iv.) The passage of a bougie, or flexible stomach-tube, is possible with a little steady pressure, and with the patient under chloroform it is easily done.   This procedure generally results in curing the condition, at any rate for a time.   (v.) The patient is most frequently of the female sex, and often presents other evidences of hysteria.   It certainly occurs also in males, and gout or rheumatism are said to predispose to it.

§ 184. **Foreign Bodies, Acute Œsophagitis, and Simple Ulcer.**—The symptoms of these conditions are much alike.   Acute œsophagitis occurs after traumatism, as after swallowing corrosive fluids,[1] or in a localised form from the presence of foreign bodies.   It sometimes occurs in the course of the specific fevers, and in infants at the breast from unknown causes.   A slighter degree of *localised* inflammation arises by no means infrequently when a fish-bone, needle, pin, bristle of a toothbrush, or other solid particle, sticks in the folds of the œsophagus.   This dysphagia takes the form of a difficulty and pain during the act of swallowing, at one particular spot.   The symptoms here start suddenly and reach a maximum at once.   This source of trouble is very apt to be overlooked when the patient has forgotten the incident which led to the lodgment of the foreign body.   When the inflammation is *generalised*, there is great pain, with consequent spasm and regurgitation on attempting to swallow.   Thirst and, if the condition be severe, feverishness are present.   Mucus, pus, and blood may be vomited if ulceration ensue.

Simple Ulcer of the gullet is very rare.   It is sometimes due to syphilis. Acute pain and tenderness are prominent features, with spasm on swallowing or on attempting to pass a bougie.   But the affection cannot be diagnosed with certainty.

· We now turn to the rarer causes of Dysphagia.

· § 185. **Paralysis of the Gullet.**—Paralysis of the upper part of the gullet—*i.e.*, of the *pharyngeal constrictors*—is not uncommon as an accompaniment and complication of diphtheria.   Difficulty of swallowing under these circumstances may be one of the first evidences of diphtheritic paralysis.   It also occurs in Bulbar Paralysis, and at the end of some slowly progressive exhausting diseases.   All these differ from the other causes of dysphagia by being attended by regurgitation of fluids through the nose.

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[1] Fluids which are simply irritating, such as beer contaminated by the substances used to clean the pewter pots, may cause the condition.

owing to the paralysis of the soft palate. Paralysis of the gullet *below the pharynx* is a much rarer condition. It may sometimes accompany and be due to the same causes as the above. It also arises as an occasional complication of general paralysis of the insane, cerebral tumour, diseases of the nuclei in the medulla, and lesions of the vagus. The dysphagia in these cases is not absolute, the normal œsophagal sound on auscultation is absent, and a *bougie passes* without hindrance. The condition can only be distinguished from simple dilatation when there is no regurgitation or pseudo-emesis of food.

§ 186. **Dilatation** or **Diverticulum of the Gullet** is not frequent, and the causes are obscure. It may take the form of (*a*) *generalised dilatation* of the whole tube ; (*b*) a *fusiform dilatation* above a stricture. (*c*) It may occur as a *diverticulum*, or sac, which is said to be formed in one of two ways : (i.) A *pressure* diverticulum or saccule, due either to weakness of the wall after injury, or sometimes to congenital weakness of some part of the tube, with consequent hernia of the mucous through the muscular wall ; and (ii.) a *traction* diverticulum, due either to adhesions between the œsophagus and neighbouring glands, or other structures, pulling out the œsophageal wall as they contract.

(*a*) A general dilatation has but few or no symptoms. (*b*) The symptoms of form (*b*) are masked by those of the stricture below. The occurrence of dilatation (with stricture) is evidenced by the regurgitation of food at shorter or longer intervals. On this account such cases are very apt to be mistaken for the vomiting of pyloric obstruction (*vide* feature 2, § 177). There is an unusual mobility in the bougie just before it reaches the obstruction. (*c*) The diverticular varieties are very rare, but, as far as we know, their symptoms are as follows : (i.) There is regurgitation of food after an interval varying from a few minutes to a few hours after ingestion. It is apt to be mistaken for persistent vomiting, but the ease with which the food is returned, and the absence of acid in it, should make us suspect this condition. (ii.) The regurgitation gradually increases in amount, and the breath is foul from the decomposition of food in the gradually enlarging pouch. (iii.) In cases of pressure diverticula a bougie which could not be passed before can be passed after vomiting, because the sac full of food forms a swelling that presses upon the gullet and so leads to obstruction. (iv.) Sometimes the pouch forms a definite tumour in the neck.

§ 187. PROGNOSIS AND TREATMENT OF DYSPHAGIA.—Dysphagia is in most cases a symptom of considerable gravity, and in severe cases it commonly enough results in death by starvation. Of all causes, malignant stricture is the most serious, and, in spite of the means which modern surgery has placed at our disposal, patients rarely live more than a year or eighteen months. The length of time depends on the maintenance of the nutrition of the individual. Next in order of gravity come tumours pressing on the œsophagus, when the prognosis depends on the nature of the tumour and its amenability to treatment.

Patients with simple stricture, and with dilatation, may live for many years on fluid diet, with or without gastrostomy, but diverticula are much more serious. Of all cases functional spasm is the most curable, although it is apt to return.

The cause of death in dysphagia is usually starvation or a low form of pneumonia. This may arise from perforation into the bronchus or by the food passing into the glottis. In either case death is expedited by the lowered vitality of the patient. Perforation may occur in other directions—*e.g.*, a case of malignant disease of the gullet under my care died from hæmorrhage consequent upon perforation into the aorta.

TREATMENT OF DYSPHAGIA.—The indications are to remove the cause of the obstruction, to maintain the strength and nutrition of the patient, and to relieve any concurrent symptoms. The question of three surgical procedures may arise in these cases : the passage of bougies of different sizes, the use of Symonds' tubes, and gastrostomy. If possible, a bougie should be passed in all cases, not only for purposes of diagnosis, but also as part of the treatment. It may be of little use in malignant stricture, but simple stricture may be dilated or prevented from further contracture by this method. Symonds' tube, a funnel-shaped tube with a string attached to prevent it slipping down, changed every three weeks or so, undoubtedly prolongs life both in malignant and advanced simple strictures. An early gastrostomy offers the best chance of prolonging life in every case of malignant stricture. In malignant stricture if, when the case comes under treatment, debility is very marked, complications are present, and there are evidences of cancer elsewhere, gastrostomy is the only treatment of any avail. Radium is now employed with success in early cases. In addition to the above treatment, the only indication in *malignant* stricture is to soothe the pain by morphia, opium, or cocaine.

In *simple* stricture, bougies of gradually increasing size should be passed and left in for some hours at a time. Force must not be used in so doing. In very narrow strictures a Symonds' tube would be better. If syphilis be suspected as the cause, potassium iodide must be given. In *functional* spasm a bougie should be passed, and cold douches given along the neck and the spine. The general condition must be treated. Valerian is given in hysteria, combined with special diet in cases with gastritis. Electricity may be useful. In *paralysis* and *dilatation*, especially the diverticular type of dilatation, the patient must be fed by a stomach-tube. If the diverticulum is high up in the neck, the surgeon may be able to remedy it. In *acute œsophagitis* the pain must be soothed by morphia hypodermically, by cocaine or morphia and ipecacuanha lozenges, or by opium given with tragacanth. Thirst may be allayed with spoonfuls of iced water, in which small doses of opium, cocaine, and milk may be administered. During the acute stage the patient may require nutrient enemata. *Foreign bodies* in the gullet need prompt attention, else they may pierce the tube and injure the aorta or other structures. With the aid of the œsophagoscope, they may be readily removed.

Feeding by a stomach-tube is a measure available in a fair proportion of cases, especially in Causes I., III., IV., VI and VII (*supra*). The only apparatus necessary consists of a long flexible rubber tube (5 feet long) one end of which is blunt, with the " eye " at the side or the end (according to choice) and the other end tied to a funnel. The method of passing the tube is either the same as that used in passing a bougie (§ 178), or it is passed through the nose. In the latter case a smaller size must be used. The operator then pours into the funnel from a jug fluid food such as milk, and mixtures of egg, milk, and malt.

CHAPTER IX

THE ABDOMEN

THE abdomen contains a large number of very important organs and structures, but just as their physiology and pathology are in many instances obscure, so also are the means at our disposal for théir thorough clinical investigation imperfect. However, it is in this region that we have to deal with symptoms which on the one hand may be of quite a trivial order, or on the other may be of extreme gravity ; symptoms and conditions the issue of which will largely depend on the promptitude, knowledge, and skill of the medical man in attendance and upon his adequate comprehension of their true meaning.

PART A. SYMPTOMATOLOGY

§ 188. Local Symptoms.—The symptoms referable to disease situated within the abdominal cavity are necessarily of the widest and most varied kind, but there are only three which are sufficiently constant to be regarded as cardinal symptoms, all of which are referable to the abdomen itself— viz., ABDOMINAL PAIN, GENERALISED ENLARGEMENT, and LOCALISED TUMOUR.

VOMITING is a fairly constant accompaniment of all acute abdominal conditions, whether the stomach is involved in the lesion or not. Its causes are discussed in § 215.

The presence of DIARRHŒA and CONSTIPATION depends very largely on whether the intestinal canal is affected, and these are fully dealt with in Chapter XI. The other symptoms also depend largely upon which of the abdominal organs is affected, with one important exception—viz., " INDIGESTION." In all chronic abdominal disorders, no matter which organ is affected, we are often consulted for " INDIGESTION " ; in fact, nausea and all the other symptoms of pronounced dyspepsia may be due to disease quite unconnected with the stomach, and located, for instance, within the uterus, kidneys, liver, pancreas or other organs. Some cases of " dyspepsia," after resisting treatment for months or years, have been cured by the stitching up of a dislocated kidney.

ABDOMINAL PAIN, if acute and sudden, is a medical emergency of the most important kind ; if chronic, it presents many difficult questions for

diagnosis. It will therefore merit the most careful study and analysis
(§ 192). The diseases *outside the abdomen* which may give rise to it are :

1. *Diaphragmatic pleurisy,* or a basal pleuro-pneumonia, may give rise
to acute abdominal pain of sudden onset (often referred to the correspond-
ing iliac region), and to abdominal rigidity and other symptoms of acute
peritonitis, which can only be differentiated by the pulse-respiration ratio
and the concurrent symptoms. *Pericarditis* sometimes causes severe
abdominal pain.

2. *Neuralgia* of the intercostal and other spinal nerves may be referred
to the abdomen. In this way spinal caries, especially in children, the
crises of locomotor ataxy, and other diseases of the vertebræ or cord, may
be mistaken for various abdominal diseases.

3. An *abscess* in the abdominal wall, a bruise, or a ruptured muscle
may be similarly mistaken, but these should present no difficulty.
Fibrositis of the abdominal wall has led to mistaken diagnoses of appen-
dicitis and ovaritis.

4. *Diabetic coma* is occasionally heralded by pain simulating appendi-
citis.

5. *Paroxysmal tachycardia* has been mistaken for a condition requiring
laparotomy (§ 59).

ABDOMINAL ENLARGEMENT and ABDOMINAL TUMOUR are considered
in Part C.

§ 189. The **General** or **Remote Symptoms** met with in abdominal dis-
orders are, as just mentioned, of an extremely varied nature, and our
endeavour should be to associate correctly these symptoms with the
abdominal organ which is affected.

COLLAPSE AND PULSE-TEMPERATURE RATIO.—In connection with the
general symptoms of abdominal diseases, one fact needs special mention -
(1) the profound collapse which is so apt to occur at the onset of acute
abdominal conditions. A blow on the abdomen may result in fatal col-
lapse, and so also may perforative peritonitis. This tendency to collapse
possibly finds an explanation in the fact that the chief centre of the
sympathetic is situated within the abdomen. Now, a subnormal tem-
perature is one of the symptoms of collapse, and for this reason it is
often present in the early stage of abdominal trouble, and it rarely ranges
very high even in the gravest abdominal conditions. In acute peritonitis,
for instance, an extensive inflammatory process affects the peritoneum,
which acting alone might produce a temperature of 105° F. or more, but
by reason of the collapse it is rarely more than 102° or 103° F. (2) *In
the pulse, however, we find our best guide to the severity of mischief within
the abdomen.* In all acute diseases, other than abdominal, we find a
rough general proportion between the height of the temperature and the
rate of the pulse. Thus, a temperature of 100° F. will correspond roughly
with a pulse of 100, 101° with 110, 102° with 120, 103° with 130, and so
on—an increase of about 10 for every 1° F. But in acute abdominal
conditions this is not so. The pulse-temperature ratio is disturbed, for

although the pulse rate increases with the severity of the abdominal mischief, the temperature never increases proportionately. Indeed, in many of the worst cases, the temperature is one or more degrees below normal. The pulse, however, is an almost infallible guide and one may say (1) that if the pulse remains under 100 nothing very serious is happening within the abdomen; and (2) that the rate of the pulse and the pulse-temperature ratio are great aids to the diagnosis, and in some sense measures, of acute abdominal disorder, especially when that disorder has reference to the peritoneum.

PART B. PHYSICAL EXAMINATION

§ 190. In the examination of the abdomen we must proceed systematically, as in the examination of the thorax, by INSPECTION, PALPATION, PERCUSSION, MENSURATION, and occasionally auscultation; though of all these measures palpation by the educated hand is at the present time the most valuable means we have. X-RAYS assist in certain cases, but this method is not always available.

1. **Careful inspection of the abdomen** should on no account be omitted; much can be learned in this way. The best point of view is that from the foot of the bed, or by bending over the patient's feet, so as to view the abdomen from below. The mere fact of enlargement may thus be verified, and whether the enlargement be generalised and uniform, or whether it be localised or asymmetrical. Notice whether the umbilicus is centrally situated, and also whether the surface presents dilated veins, such as occur in abdominal cancer, or when the portal vein or vena cava is obstructed. Dilatation of the abdominal veins is met with chiefly in three conditions: (1) In liver cirrhosis, these veins being part of the conservative collateral circulation which gradually becomes established; (2) the veins without being much dilated or prominent are *unduly apparent* in cases of abdominal carcinoma. It is a sign of considerable value and constancy. (3) Extreme dilatation and varicosity of the superficial veins occurs only when the inferior vena cava is obstructed. This is generally due to a gummatous deposit in or around the posterior border of the liver where the vena cava passes through it. The veins of the legs and testes generally share to a less extent in the dilatation.[1] Notice also whether there is any fistula, thickening or infiltration round the umbilicus such as may occur in cancer and tuberculous peritonitis. An abdominal enlargement due to the presence of air or gas is rounded anteriorly, but when due to fluid it is usually flattened in front and the flanks bulge; when there is obstruction of the large intestine the flanks bulge; whereas in obstruction of the small intestine low down the swelling occupies the centre of the abdomen. Incidentally you may notice the presence or absence of the white lines (lineæ albicantes) left by a previous pregnancy,

[1] A case is recorded by Dr. W. Chapman, Clin. Soc. Trans., 1899 and 1900, and *Lancet*, December 2, 1899.

the knowledge of which may be medically useful. The presence of hernia
or of tumours of the wall may be recognised by inspection. The amount
of movement of the abdominal wall with inspiration should be noticed,
for diminished or absent movement constitutes an important sign of peri-
tonitis. If the peritonitis is local, the abdominal wall over that area may
move badly, whilst elsewhere abdominal respiratory movement is normal.
Pulsation seen in the epigastrium is not necessarily abnormal, but may
be due to the right ventricle or an engorged liver secondary to heart
failure. Sometimes aortic pulsation is unduly visible, especially in neurotic
dyspeptic women, or it may be transmitted by a pyloric tumour. Rarely
the pulsation is due to an abdominal aneurysm. Visible peristalsis should
also be looked for, and if present its position and direction should be
noted.

The REGIONAL ANATOMY OF THE ABDOMEN is important as a guide to
the seat of disease (Fig. 59).

2. PALPATION.—Considerable experience is necessary for satisfactory
abdominal palpation. The hand should be warmed and always laid *flat*
on the abdominal wall; then by gently dipping in the fingers, by flexing
the metacarpo-phalangeal joints, we have the most ready method of
ascertaining (1) the presence of any tumour; (2) the boundaries of some
of the solid organs. The patient should lie on his back with the knees
drawn up and the shoulders somewhat raised, so as to relax the abdominal
muscles. Do not use the tips, but only the pads of the fingers, for the
tips stimulate the recti muscles to contract, and thus to simulate a tumour
where none exists. Many patients offer considerable involuntary or
voluntary resistance, and this must be overcome by placing them in an
easy posture and distracting their attention, or asking them " to let the
breath go." Relaxation may be obtained by immersing the patient in
a hot bath; in some cases it may be necessary to use chloroform. Much
obesity is another obstacle to palpation. Palpation reveals the presence
of localised resistance and tenderness which denote underlying inflam-
mation, but it must be remembered that in severe toxæmia this reflex
rigidity may be very slight. Tumours, flatulence, and the movement
of fluid within the abdomen can also be detected by palpation. The
palpation and percussion boundaries of the different organs are described
in later chapters.

3. PERCUSSION of the abdomen is carried out with the same precautions
as in the case of heart and lungs, and the student will now find it very
convenient to be able to percuss with either hand indifferently. The
liver and spleen are the organs which give a dull note on percussion. By
this means we ascertain the presence of solid and fluid, which are dull,
or of gas, which is resonant. When the fluid is free the dulness alters
with the position of the patient.

4. By MEASUREMENT we ascertain the amount of increase in size. As
a general rule, horizontal measurement should be taken at the level of
the umbilicus, and it should be recorded for future reference. In order

6. X-RAY EXAMINATION may give much assistance in obscure and in chronic cases.

The FALLACIES of abdominal enlargement are : (1) *Fat in the omentum* is referred to under fluid enlargement (§ 209). (2) *Phantom tumour* is described under abdominal enlargement due to gas (§ 207). (3) *Pendulous abdomen*, so frequent in elderly women, is often thought by the patient to be a " tumour," but it is due only to weakness of the muscles of the abdomen and of the intestinal tube. (4) *Pregnancy* and distended bladder are frequent causes of error (§ 212). (3) In *rachitic children* the liver and spleen may be pushed down by the deformity of the costal arches, and so produce the appearance of an enlarged abdomen. (6) Apparent enlargement of the abdomen may be caused by the pressure of some thoracic tumour. (7) The most frequent cause of abdominal enlargement is the presence of venous congestion and gas in the intestine.

PART C. ABDOMINAL DISORDERS : THEIR DIAGNOSIS, PROGNOSIS, AND TREATMENT

§ 191. Routine Procedure and Classification.—Having *first* ascertained that the patient's leading symptom is one of those above referred to, we *secondly* inquire into the history, and especially whether the condition came on acutely and suddenly, or is chronic and long-standing. The procedure to be adopted in acute cases, and in chronic cases, is given under their respective headings. *Thirdly*, proceed to the physical examination of the abdomen, the routine method in ordinary cases consisting of (1) Inspection ; (2) Palpation ; (3) Percussion, to map out the boundaries of the liver, spleen, and other organs ; and (4) Mensuration. In any doubtful case the rectum, vagina, hernial orifices, urine, and fæces must be examined. The fallacies mentioned in § 190 must be borne in mind.

If **severe abdominal pain,** which came on **suddenly** and acutely, be the leading symptom, first turn to § 192.

If **abdominal pain** of some duration and running a **chronic** course be the leading symptom, turn to § 198.

If there be a **generalised abdominal enlargement,** turn to § 206.

If there be **localised tumour,** turn to § 211.

§ 192. Acute Abdominal Pain, coming on **suddenly,** includes amongst its causes some of the most serious conditions with which a physician or surgeon can have to deal ; and on account of the large number of organs contained in the abdominal cavity, these causes include many pathological processes situated in various and often unsuspected positions.

The *causes* of abdominal pain may be conveniently classified for clinical purposes into nine groups :

A. ABDOMINAL PAIN coming on **suddenly, with collapse.**

    I. Perforation of some organ or cyst (perforative peritonitis)   § 193
    II. Acute peritonitis due to causes other than the preceding   § 194

In the first six the acute abdominal pain is usually ATTENDED BY COLLAPSE, but not in the last three. This, however, is only relative, and in any doubtful case the whole should be passed in review.

In order to ascertain which of these causes is in operation, and in view of the gravity of some of these cases, it will be desirable to consider the METHOD OF PROCEDURE in some detail.   •

(1) Regarding the *cardinal* or *leading* symptoms, inquire carefully, as in all cases of "pain," concerning its position, character, degree and intensity. The position of the pain is not always a guide to the organ affected, for it rapidly tends to become generalised; but the direction in which it is radiated is of great help in the diagnosis of the three kinds of colic. Moreover, local disease may be accompanied by generalised pain, and widespread disease may give rise to a localised pain. Whenever the three symptoms—ABDOMINAL PAIN, VOMITING, and CONSTIPATION—come on together suddenly, with COLLAPSE, the condition is very probably due to either PERITONITIS (which may be due to PERFORATION), or INTESTINAL OBSTRUCTION.

2. As to the *History of the Illness*, it is useful to note if there had been any illness previous to the onset of the pain pointing to ulceration, dyspepsia, or other derangement of the abdominal organs. The occupation may shed some light on the cause- *-e.g.*, sudden strain, working with lead. The description of the mode of onset may assist—*e.g.*, "something was felt to give way."

3. In the *Examination of the Patient*—(i.) his *age* is an important aid in the diagnosis of the cause of the pain. In childhood it is very probably some intestinal affection, such as colic, or intussusception; in adolescents and young adults, appendicitis, while cancer and tabetic crises may probably be excluded. In adults we think of hernia and ulcer of the stomach; in old age and after middle life we think of cancer, or if the patient is a female, biliary colic. (ii.) The *sex* may aid us, for in young females we may suspect an ulcer of the stomach even without previous symptoms; and in older women the rupture of an ectopic (extra-uterine) pregnancy, frequently overlooked, or gall-stones. (iii.) The presence of rigidity, as shown by resistance to palpation, or of *tenderness* is of considerable aid; they point to the existence of underlying inflammation. (iv.) *All the organs* of the abdomen must be as carefully and as thoroughly examined as circumstances will permit. Never forget to examine per rectum and

vagina, because stricture of the former or a pelvic abscess or tumour may throw considerable light upon the case. (v.) The patient's *general symptoms* must also be carefully investigated. If the temperature *and the pulse* be normal, we may exclude inflammatory conditions. The temperature alone is not a sufficient guide in this respect (see § 189), but in general terms no serious acute abdominal condition exists without the *pulse rate* exceeding 90 or 100. If the patient is much emaciated, in adults we must bear in mind obscure malignant disease, and in children the presence of tubercle. Examine the urine for sugar, and do not forget to examine the chest (see § 188).

If the pain, which is severe and has come on suddenly, is **attended by marked collapse**, first turn to § 193. If it is **unattended by collapse**, turn first to § 196. It must be remembered, however, that any severe pain will cause a certain amount of prostration.

I. *The patient complains of* **acute abdominal pain,** *which has come on* **suddenly,** *with symptoms of severe* **collapse,** *attended by* vomiting *and* CONSTIPATION ; *the pulse is rapid (over* 100). *The case is probably one of three conditions,* PERFORATION *into the peritoneum,* ACUTE PERITONITIS, *or* ACUTE INTESTINAL OBSTRUCTION.

§ 193. **Rupture of a Cyst, Abscess, or Organ, or Perforation of the Alimentary Canal** (which shortly develops into Perforative Peritonitis). The *cysts which may rupture* are hydatid or simple cysts of the liver, kidney, pancreas, or other organs, ovarian and parovarian cysts, and abscesses of the liver, gall-bladder, kidney or other organs, or of mesenteric glands and perityphlitic abscess (§ 199). *Rupture of an Organ,* with consequent extravasation of blood, causes similar symptoms. The most common are : ruptured Fallopian tube (in cases of extra-uterine pregnancy), ruptured abdominal aneurysm, rupture (following injury) of the liver, kidney or spleen, a ruptured abscess of these organs. *Perforation* of the alimentary canal may at any time occur when an ulcer is present. These ulcers are, simple ulcer of the stomach (which is usually met with in young anæmic women), simple ulcer of the duodenum (which occurs in males), ulcer of the lower part of the ileum (due to tuberculosis or fever), ulcer of the cæcum or appendix, ulcer of the large intestine, especially the sigmoid flexure (usually cancerous, dysenteric, or syphilitic).

Symptoms.—Pyrexia at first is absent, and the temperature may be subnormal because of the collapse. The pulse, at first normal, becomes thready, feeble, and rapid. The pain is probably very severe, and the ashen pallid face, with its cold, clammy sweat and sunken eyes, is very characteristic. Vomiting is rarely absent ; it may be incessant, distressing, and even stercoraceous. A certain amount of constipation is generally present on account of the paralysis of the bowel. Perforated *gastric ulcer* is perhaps the commonest of the conditions above mentioned, and may be taken as a type. We should inquire for a history of dyspepsia and other symptoms (§ 231), but in not a few cases rupture has occurred without

previous symptoms of any kind whatever. On examination there is tenderness, rigidity of the muscles most marked in the epigastrium, and a tympanitic note over the whole abdomen. The disappearance of the liver dulness on the mid-axillary line denotes free gas, and is usually due to ruptured gastric ulcer. After a few hours there is a deceptive latent period during which all symptoms of discomfort are diminished. A *stage of reaction* occurs several hours later, when symptoms of collapse are found, with acute peritonitis (§ 194), generalised or localized. There is increased abdominal distension, vomiting and tenderness with decreased rigidity. The symptoms of perforated *duodenal ulcer* resemble those of gastric ulcer, but the condition occurs usually in men. The symptoms of perforation of another part of the intestine, or rupture of a cyst, are much the same, and one can only hazard a diagnosis as to its situation by the site of the pain and tenderness, and the previous history. Three degrees of severity occur with perforation of the intestine : (*a*) When there are adhesions the peritonitis may be localised or partial ; (*b*) when there are no adhesions, but a small leakage, it may be only moderately sudden in its onset ; (*c*) when the leakage is large it is extremely sudden and severe in its onset.

The *latent period* which ensues shortly after an acute onset deceives many clinical observers. The pain may subside, all symptoms decrease, and the temperature become normal or subnormal. But (1) the *pulse rate remains persistently high*, and (2) in the blood there is marked and progressive leucocytosis. These are sufficient to indicate immediate exploratory abdominal section. Perforative peritonitis may have to be diagnosed from diaphragmatic pleurisy and pneumonia of the base, in which the pulse-respiration ratio is disturbed, but not the pulse-temperature ratio.

Treatment and Prognosis.—Laparotomy should be performed at once. If the period of repose leads one to believe the patient is recovering, in a few hours general peritonitis will have set in, and operative interference will be too late. In cases where patients have been operated upon within the first twelve hours 79 per cent. have recovered ; if after twenty-four hours only 29·4 per cent. have recovered (Goffe). The after-treatment depends on the cause. In the case of rupture consequent on injury internal hæmorrhage may take place with a rapidly fatal result, but even in such cases early laparotomy has been performed with success.

II. *The patient complains of severe* abdominal pain, *extreme* prostration, *and* VOMITING ; *there is* THORACIC RESPIRATION, *and the* TEMPERATURE *is* ELEVATED. *The disease is* ACUTE PERITONITIS.

§ 194. Acute Peritonitis (General Peritonitis) is an acute inflammation of the peritoneum. It is rarely a primary disease, but its onset is usually sudden.

Symptoms.—(1) The aspect is very characteristic ; the countenance has an anxious pinched look, the cheeks flushed, and the skin cold and

clammy. (2) The pain is severe and constant, but liable to exacerbations on account of the intestinal peristalsis and the passage of wind along the bowel.[1] It is also increased by any kind of movement, even by the respiratory movements. Consequently (3) the respiration is thoracic, and careful inspection will show that (4) the abdominal walls are immobile and rigid. There is acute tenderness on pressure, so much so that the weight of the bed-clothes can hardly be borne. (5) The posture of the patient is very characteristic as he lies on his back with legs drawn up to relax the abdominal muscles. (6) Pyrexia, often ushered in with sudden rigors, and attended by a small, wiry, rapid pulse of 100 to 140 per minute. The temperature is elevated only 2° or 3° F. above normal, and maintained there continuously, unless pyæmia be present, in which case there are rapid variations of wide range. In some cases—*e.g.*, perforation—it may be subnormal at first (*vide supra*). There is marked prostration, as in all abdominal inflammations, and a great tendency to collapse, even from the beginning. (7) The bowels are constipated, and there is persistent vomiting. Hiccough is often present, and if persistent is a very bad sign, as in all abdominal disorders. There is diminution of urine—which may amount to suppression. The urine is abundantly charged with indican. Death occurs from collapse or asthenia, and the mind remains quite clear until the end in uncomplicated cases. Peritonitis is seldom a primary affection, and careful inquiry should reveal the cause.

In acute localised peritonitis the symptoms are those of acute general peritonitis, but are less severe, and are more confined to the affected region.

The *Causes* of acute peritonitis may be grouped under seven headings : (i.) *Injury or Operation.*—In cases occurring in women without obvious cause, the possibility of criminal procedure for abortion should always be remembered. As regards surgical operations on the belly, modern experience has shown that it is not the actual injury but the introduction of septic organisms, which produces the peritonitis, and that if these be excluded mere damage to the peritonæum will not cause a generalised peritonitis. (ii.) *Extension* of inflammation from the thorax, or from various organs of the abdomen—*e.g.*, appendicitis, gonorrhœal salpingitis, inflammatory conditions of the intestines (typhoid, dysenteric, and other). (iii.) *Blood Infections* of various kinds—*e.g.*, pneumococcal, streptococcal staphylococcal, and gonorrhœal. *Idiopathic Peritonitis* was the name formerly employed when no cause could be discovered. Peritonitis is apt also to complicate scarlatina, dysentery, and the other acute specific fevers. *Puerperal Peritonitis* arises when septic organisms enter through the raw uterine surface. A chronic form of the disease arises in *uræmia*. *Bacillus coli communis* may produce peritonitis either as part of a general septicæmia, or primarily. (iv.) *Chill*, under certain conditions, such as bathing during the menstrual period, is sometimes included as a cause, though this usually leads to a chronic localised peritonitis (perimetritis).

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[1] The acute peritonitis which complicates typhoid fever is of a latent character, and unaccompanied by pain. This and puerperal peritonitis are the only exceptions.

The true explanation of peritonitis after a " chill ". is to be found in microbic infection. (v.) *Rupture* of an organ or some abdominal cyst, such as ovarian cyst, or an abscess of the liver, or rupture of the gall-bladder, etc. (§ 193). (vi.) *Perforation of some part of the alimentary canal,* which had previously become thin by ulceration—ulceration of the appendix vermiformis, simple ulcer of the stomach or duodenum (malignant ulcer rarely or never perforates because of the infiltration around), typhoid or tuberculous ulcer of the ileum, etc. (see Perforative Peritonitis). (vii.) Any condition such as volvulus or intussusception, in which the resistance of the intestinal wall to the passage of organisms is diminished, may be a cause of peritonitis, local or general.

Acute general peritonitis has to be *Diagnosed* from four diseases : (1) Acute intestinal obstruction, in which the constipation is absolute and no flatus is passed ; there is usually no pyrexia, and the constitutional disturbance is usually less. (2) In *colic,* although the pain is also very severe, there is an absence of tenderness, and pressure may give relief. Pyrexia and collapse are absent, and the pulse is normal. (3) In *catarrhal enteritis* there is pain, and there may be vomiting and tenderness on pressure, but in this disease there is profuse diarrhœa. (4) In certain cases of *hysteria,* acute peritonitis may be very accurately simulated, though the temperature and pulse are normal, there is very little collapse, and evidences of the hysterical diathesis are present.

The *Prognosis* of general peritonitis is always very serious. As regards etiology, perforative peritonitis, formerly considered the gravest, is probably now the most hopeful if promptly dealt with. Modern surgery has done much for the rescue of such cases, and undoubtedly the most favourable of them is that due to appendicitis. Cases of this disease, if properly managed, should hardly ever be lost. The prognosis in any particular case depends therefore on the time elapsing before operation, and secondarily on the cause and the severity of the collapse, the dyspnœa, and the hiccough.

Treatment.—The treatment of acute peritonitis depends upon whether it is general or local. If *general,* the only rational treatment is by operation immediately a diagnosis has been made. A fatal issue is almost invariable in cases not operated upon, since the condition is rarely primary, and a definite local lesion is usually present. If for any reason an operation cannot be done, recourse must be had to the older methods of treatment. In *local* peritonitis medical treatment is indicated, but even in this condition, if there are signs which make it probable that pus has formed, an exploratory incision should be made. Medical treatment comprises keeping the patient in bed and relieving symptoms. The diet should be fluid, consisting of soups, jelly, milk, to which stimulants may be added according to the condition of the pulse. Rectal feeding may be necessary. Local applications may give relief, especially cold in the form of icebags, or ten or twelve leeches to the abdomen. Fomentations, either simple or with tincture of belladonna, relieve the pain. The most valuable

drug is opium, for it relieves the pain, and reduces the peristalsis of the bowel, and so gives local rest. It may often be given in fluid form by the mouth, and can be tolerated in large doses. If vomiting persists it should be administered hypodermically. If there is any doubt as to the advisability of a surgical operation, either immediately or later, opium should be withheld, for by masking the symptoms it may lead to a continuation of medical treatment when operation is called for. It is therefore of use chiefly in local peritonitis, or in general peritonitis where an operation is not permitted. Purgatives are better avoided, but the lower bowel should be opened by means of enemata. The hiccough may be relieved by giving ice to suck, and by opium or chloral.

III. *The patient complains of* **acute abdominal pain** *which is* **attended** *by* **collapse,** *and the* PULSE IS RAPID ; *there is* ABSOLUTE CONSTIPATION, *with inability to pass even flatus, and* VOMITING (*at first of food, then of bile, and finally of stercoraceous matter*)—the condition is ACUTE INTESTINAL OBSTRUCTION.

Acute Intestinal Obstruction—*i.e.,* obstruction coming on suddenly, is always a matter of serious importance, and every practitioner should be thoroughly acquainted with its several causes. In actual practice, whenever the three symptoms, **constipation, vomiting,** and **abdominal pain** occur together, one of three conditions should be suspected—acute peritonitis, intestinal obstruction, or colic.

The various causes of acute intestinal obstruction—the chief of which are External Hernia, Internal Strangulation, and Intussusception--are fully dealt with under Intestinal Disorders (Chapter XI). **Appendicitis** is mentioned by some as a cause of intestinal obstruction. Appendicitis may first present itself as an acute disease ; it is described in § 199.

§ 195. *The patient complains of* **acute abdominal pain,** *with more or less* **collapse** ; *the temperature is probably normal or subnormal, but the symptoms do not quite conform to any of the preceding*—some of the **rarer causes** are probably in operation, such as the following :

IV. **Displacement of a Gravid Uterus** is known by the pain being referred to the pelvis, and examination revealing the local mischief. It may occur when jumping from a height, and performing active exercise, especially in early pregnancy (Chapter XIV).

V. In **Embolism of the Mesenteric Artery,** a cause of embolism, such as endocarditis is present. It is rarely diagnosed during life. The absence of symptoms pointing to the other causes may lead one to suspect embolism. Embolism of the spleen may also cause severe symptoms.

VI. **Acute Pancreatitis.**—(1) The pain here is very sudden and severe, usually in the upper part of the left side of the abdomen and extending to the lumbar region ; (2) constipation and severe vomiting (never fæcal) are usually present ; and (3) there is usually tympanitic abdominal distension (see also § 205) with rigidity and epigastric tenderness ; (4) cyanosis, when present, aids the diagnosis.

Hæmorrhage into the Pancreas is attended by : (1) severe and sudden pain in the upper part of the abdomen, and that part soon becomes tender ; (2) vomiting of increasing severity ; (3) symptoms of collapse, with the restlessness and subnormal temperature which accompany collapse when it is due to hæmorrhage (§ 205).

The *Diagnosis* of both of these conditions from intestinal obstruction or perforation into the peritoneum is usually impossible before laparotomy. In both diseases death from collapse is usual.

Acute inflammation (**adrenalitis**) or hæmorrhage into the suprarenal capsules produces symptoms similar to those of acute pancreatitis. There is sudden abdominal (epigastric) pain, with vomiting and collapse. Death may occur in a few days. Or there may be convulsions and coma, or extreme muscular weakness for some days before death. It is rarely diagnosed during life.

VII. *The patient, while apparently in good health, complains of* acute abdominal *pain, which has come on* suddenly, without *definite* collapse ; *the pulse does* NOT EXCEED 100; *there may be* VOMITING *and* constipation. The case is probably one of the three kinds of COLIC, though APPENDICITIS, VISCERAL NEURALGIÆ, and some OTHER AFFECTIONS may start in this way.

§ 196. **Colic is** a somewhat vague term applied to spasmodic paroxysmal pain situated in the abdomen. There are three kinds—**intestinal, hepatic, and renal colic**—and they have the following features in common : (1) The pain is extremely severe, and sudden in its onset ; (2) not infrequently there is vomiting from the severity of the pain ; (3) the face is pale and " anxious," and in severe cases the pulse is rapid and feeble, though it practically never exceeds 100 ; (4) *the temperature is neither above nor below normal;* (5) the physical signs in the abdomen are negative, and the pain may even be relieved by pressure ; (6) the patient is " doubled up " with pain, restless, trying to find a position of comfort. In intestinal colic a hardening of the bowel may be appreciated by the palpating hand.

(*a*) **Intestinal Colic** is due to distension and spasm of the bowel. The colic of the small intestine is characteristically twisting, paroxysmal, and is referred to the epigastrium or umbilicus ; colic of the colon is referred to the hypogastrium. Intestinal colic is relieved by pressure, which distinguishes it from peritonitis. The abdomen may be distended with flatus. Sometimes it is followed or accompanied by diarrhœa, or, as in lead colic, by constipation. Colic may be the first sign of lead-poisoning, or may be accompanied by a slow, hard pulse, with other symptoms of plumbism, such as a blue line on the gums ; and a history of working amongst lead is obtainable (§ 435).

(*b*) **In Hepatic Colic** which is due to the passage of a gall-stone into the bile duct, the pain shoots *upwards* to the right shoulder and backwards to the scapula, never downwards ; a dull pain continues during the intervals between the spasms. After lasting a few hours or a day or two it is followed by jaundice. A history of previous attacks assists the diagnosis.

(*c*) **Renal Colic** is due to the movement of a calculus, crystals or blood in the kidney or along the ureter. The pain radiates *downwards* from the loin to the thigh and the testicle of the same side, which is often retracted. It may last for a day or two. During the attack micturition is frequent ; sometimes there is hæmaturia or strangury. There will probably be a history of gravel in the urine, or attacks of a similar nature.

The *Diagnosis* of the forms of colic is given below. An X-ray examination should be made when repeated attacks of colic occur.
Prognosis.—The course of an attack of colic is short and severe.
Treatment.—For all forms of colic some of the following measures—hot fomentations, a hot bath, belladonna, turpentine, opium, or chloroform, as local applications, and hypodermics of morphia, gr. ¼ (0·01), with atropin, gr. ₆₀ (0·001)—may be necessary to alleviate the extreme pain. Large draughts of warm water should be taken. For intestinal colic in particular, a full dose of castor oil, with 20 minims (1·2) of laudanum, should be given, followed by saline purgatives. For lead-poisoning, see § 435. Hepatic colic is dealt with under gall-stone (§ 266) and renal colic in § 327.

TABLE XIII.—DIAGNOSIS OF COLIC.

	Character and Distribution of Pain.	*Associated Symptoms.*	*Age and Sex of Patient.*
Intestinal.	Twisting, around umbilicus, paroxysmal; relieved by pressure.	Constipation (or diarrhœa). No jaundice.	Any age or sex. Sometimes evidence or history of plumbism.
Biliary.	In right hypochondrium, shooting upwards to right shoulder, constant, but also in paroxysms.	Jaundice soon supervenes. Other hepatic symptoms may be present.	Female sex. At or after middle life.
Renal.	In loin, shooting down to thigh and testicle or ovary of same side.	Crystals or other urinary change, hæmaturia. No jaundice. Sometimes frequent micturition or strangury.	Usually male. Children and adults.

§ 197 VIII. Among the **rarer causes of acute abdominal pain** without collapse are various OBSCURE ORGANIC AFFECTIONS of the abdomen, evidenced at first only by pain. Two may be mentioned which came under my notice, PANCREATIC CALCULUS and OBTURATOR HERNIA, in both of which the only symptom for some time was pain coming on SUDDENLY without collapse. In the former the pain was extremely severe, and of a paroxysmal character, situated just below the umbilicus; later on it was associated with fat in the fæces, emaciation, and glycosuria.

DISLOCATED or FLOATING KIDNEY (§ 201), which is a more frequent condition than is usually supposed, may be attended by a constant (chronic) pain, or give rise to severe attacks (Dietl's crises), hardly distinguishable from intestinal colic.

APPENDICITIS is also a cause of abdominal pain, which may be of sudden onset. Sir William Macewen told me of the case of a young man who was *suddenly* seized with severe abdominal pain in jumping out of a hansom cab. But appendicitis is rarely so acute, and is therefore treated of more fully in § 199.

In SPLENIC EMBOLISM the pain is generally sudden in onset, but is not usually very severe or lasting, and is referred to the splenic region. Its most common cause is acute or chronic endocarditis, evidences of which are present.

In HENOCH'S PURPURA, and angio-neurotic œdema there may be acute recurring attacks of colic simulating intussusception. For differential features see §§ 485 and 527.

In most obscure organic affections the pain comes on gradually, and is of a chronic character. Acute pain occurring in attacks of varying duration is met with in cases of membranous or mucous COLITIS, and the VISCERAL NEURALGIÆ. DIABETIC COMA

is sometimes heralded by pain, usually in the epigastrium, which may be very severe
(§ 188). Exaggerated abdominal breathing is a useful diagnostic aid.

.. IX. In **Visceral Neuralgias** abdominal pain may come on suddenly and acutely,
and may be for a long time the only symptom.

1. *Gastralgia*, or gastric neuralgia, is rare, but it is the most typical and best-known
visceral neuralgia. The pain is severe, periodic, but usually relieved rather than
aggravated by food or by pressure. The skin may, however, be very sensitive to the
flick of a handkerchief (§ 230).

2. The gastric crises and neuralgia of the bladder or other viscera in association
with *tabes dorsalis*.

3. *Neuralgia*, or " colic " of other viscera (*i.e.*, pain in the viscus without functional
or organic derangement), has been described by various authors—*e.g.*, ovarian colic,
vesical or splenic neuralgia, etc.—though these cases sometimes turn out to be con-
nected with an undiscoverable organic disease, or with *tabes dorsalis*.

4. The neuralgia which accompanies or follows *herpes zoster*.

5. *Angina Pectoris* is in some cases referred more to the abdomen than it is to the
chest, but it is recognised by the circulatory disturbances (§ 47).

6. *Migraine* is certainly met with, alternating with abdominal pain.

§ 198. By **Chronic Abdominal Pain** I mean that kind of abdominal pain
which has come on somewhat gradually, and is running a chronic course.
Chronic abdominal pain may be produced by a large number of causes
which it would be impossible even to enumerate. It is only possible here
to refer to those conditions which do NOT PRESENT signs or symptoms
distinctly pointing to some affection of the STOMACH, LIVER, SPLEEN, or
other ABDOMINAL VISCUS. Abdominal pain is the leading or only symptom
in the following conditions :

I. Appendicitis   ..   .. § 199
II. Chronic intestinal obstruction (malignant stricture, simple stricture,
    pressure by a tumour, paralysis of the bowel, etc.)   ..   .. § 254
III. Chronic peritonitis..   ..   .. § 200
IV. Movable kidney   ..   .. § 201
V. Intestinal dyspepsia ;   VI. Enteroptosis ;   VII. Obscure visceral and
    spinal disease ;   VIII. Pancreatic disease   ..   ..   .. §§ 202-205

The history must be thoroughly investigated, and every organ
thoroughly examined. Three features may afford us important clues :

1. The POSITION, character, degree, and constancy of the *pain*, and the presence
of *tenderness* must be observed. (i.) If the pain and tenderness be *generalised*, one
may suspect Tubercle or Cancer of the Peritoneum. (ii.) If they be situated chiefly
in the *lower abdomen*, one may suspect Appendicitis or incipient disease of the Bladder
or Uterus. (iii.) If the pain be chiefly in the *upper abdomen*, incipient Gastric or
Liver disease. Thorough and REPEATED EXAMINATIONS of the *abdomen*, *rectum*, and
vagina are nearly always necessary. The *urine* also should be repeatedly examined for
blood, pus and crystals, and the *faeces* (§ 239) for gall-stones. Occult blood and
chemical changes pointing to disease of some organ may be detected by expert examin-
ation of the faeces. If there be general abdominal enlargement, turn to § 206 ; if a
localised tumour, turn to § 211.

2. The AGE of the patient, and the history and duration of the illness should be
inquired into. In *children* perhaps the commonest of the obscure causes of chronic
abdominal pain are intestinal worms and tuberculosis of the peritoneum ; in the *aged*
incipient cancer of some organ.

3. The STATE OF THE BOWELS, both previously and at the time of examination.

In L., II., and III. above there is constipation, while in most of the other causes there is diarrhœa or irregularity of the bowels.

The **Abdominal Pain** is *constant, but liable to* exacerbations, *especially after exercise ; there is* TENDERNESS *in the right iliac region ;* the PULSE *is* RAPID ; there is NAUSEA or VOMITING, *with some elevation of the* TEMPERATURE ; *the patient is young.* The disease is probably APPENDICITIS.

§ 198. **Appendicitis** is much more common than used to be suspected, yet it is still frequently overlooked, especially in chronic cases. Appendicitis may consist simply of a *catarrhal* inflammation of the vermiform appendix, which may go on to *Ulceration, Peritonitis* (usually localised), or *Perforation.* If it subsides, there is usually left some degree of inflammation which may be insufficient to cause appreciable symptoms, yet predisposes to attacks of a more acute nature. Acute appendicitis is often associated with an impaction in the appendix of intestinal concretions. Inflammation may extend to the cæcum (typhlitis) or the surrounding tissues (perityphlitis). If the lumen is blocked, as by any foreign body, an abscess forms in the tip of the appendix, with localised peritonitis. If the disease subsides at this stage, adhesions are formed which, when they contract, may kink the lumen and give rise to another attack. The diagnosis from ileo-cæcal gland disease is difficult in this stage. The inflammation may go on to ulceration and perforation, and a localised peritonitis, again with the formation of adhesions, may result. On the other hand, adhesions due to a former attack may not be dense or widely distributed enough to prevent the occurrence of a generalised peritonitis. If the localising inflammatory reaction of the peritoneum is adequate, an abscess will form, and may give rise to very few symptoms, or may gradually extend until its subsequent treatment becomes very difficult and fraught with grave danger to the patient. The results of such extension, which may be very slow or extremely rapid, are subdiaphragmatic abscess, pyosalpinx, rupture into the bowel or bladder, or externally above Poupart's ligament. It is very rarely now that cases are allowed to reach such an advanced stage that the last complication can occur. In acute cases the complications most to be feared are general peritonitis, perinephric abscess, and implication of the liver by spread along the vessels or lymphatics.

There are two clinical forms of appendicitis : Acute or recurrent, and chronic appendicitis. (*a*) In CHRONIC APPENDICITIS there may be no symptoms other than pain in the right iliac region increased after any over-exertion. Sometimes the pain is referred to another part of the abdomen ; sometimes there is also alternating diarrhœa and constipation ; there may or may not be local signs of swelling or tenderness, and a history of general malaise. One form of chronic appendicitis is due to malignant disease or tuberculosis of the cæcum or appendix.

(*b*) RECURRENT APPENDICITIS consists of recurring acute attacks. Here again the course of the disease is essentially a chronic one, and is frequently due to colitis. The patient may go on for many months in apparent

health, but in the vast majority of cases a fresh attack of inflammation occurs sooner or later. Such cases often do well with appendicostomy instead of removal.

Symptoms.—In a typical acute *attack of appendicitis* there are three symptoms, which, occurring in a young person, point to appendicitis— pain with tenderness, local resistance or swelling, and quickened pulse. (1) The chief symptom, as above mentioned, and sometimes the only one, is pain with tenderness, usually situated in the right iliac region. The *tenderness is generally fixed*, and is nearly always in this situation ; but the *pain has a tendency to radiate*, and it may be referred to the umbilical, or even to the left inguinal region. Special tenderness is present at " Mac-Burney's point "—*i.e.*, midway between the umbilicus and the right anterior superior iliac spine. In other cases the pain may be diffuse at first and later concentrate in the appendix area. (2) There is a feeling of resistance or of rigidity, or an indefinite tumour, with dulness to percussion, in the right iliac fossa. This local swelling may be due to abscess formation or to septic infiltration of the subcutaneous tissues. (3) The pulse is quickened and thready. It forms the best single indication of the acuteness of the progress of the attack. The temperature very often falls with the onset of gangrene, but the pulse, except in very rare cases, remains rapid. The temperature usually rises soon after the onset of the pain, and remains about 100° to 102° F. for a few days (Fig. 106, § 414). (4) Vomiting may be urgent at the onset of an attack ; when it continues for many days the prognosis is unfavourable. Constipation is usually present, so that the case is apt to be mistaken for intestinal obstruction ; but in some cases the attack is ushered in with diarrhœa. The urine is scanty ; the bladder irritable.

Course and Prognosis.—When an acute attack, as above described, sets in, there are three possible events—recovery, local abscess formation, or general peritonitis. (1) In a favourable case the temperature falls about the third day, the swelling disappears, pain and other symptoms subside, and the patient may be well in ten days. In other cases slight fever persists for a few weeks, and there is left an indurated swelling due to adhesions. The patient may go about for months or years with chronic appendicitis, and apart from vague pains, general malaise, and dyspeptic symptoms suffer no inconvenience. At any time, however, he is liable to have a recurrence of the acute symptoms. (2) When the general symptoms show no improvement by the third day, and the local swelling progressively increases, it is probable that an abscess is forming. (3) Perforation, with generalised peritonitis, may occur at any time. The general symptoms in such cases are much more severe, vomiting persists, and the abdomen is distended and motionless by the second or third day. There is no disease in which it is more dangerous to hazard a prognosis. An apparently convalescent case may develop general peritonitis and die within twenty-four hours ; on the other hand, a case presenting every sign of a large and extending abscess may clear up entirely and prove

free from any subsequent attack. Apart from the great improvement in the prognosis when immediate operation is performed, the only indications of value for the purpose of forming an opinion are the condition of the patient as regards shock, collapse, and age. The younger the subject, the more likely is the disease to prove fatal.

Treatment.—Rest in bed and light diet are essential. Hot fomentations locally are useful for the pain. Opium in small doses (short of causing drowsiness) is also admissible for the relief of pain after the diagnosis is established. Neither opium nor heroin should be given for long. Other hypnotics, such as omnopon and spasmalgin, may be employed.

The *question of operation* requires careful consideration, and a surgeon should be early in touch with the case. The largest proportion of recoveries is recorded in cases operated on within twenty-four hours of the onset of symptoms which enabled a diagnosis of appendicitis to be made. The subsidence of symptoms is not necessarily a contra-indication to operation. The onset of gangrene, in particular, may cause a sudden subsidence of all signs of acute disorder ; even the pulse-rate may return to within normal limits. The most valuable sign, in the absence of clinical indications, is the presence of a leucocytosis. If this goes above 20,000, or is found to be rising when two or more estimations are made at intervals, there is so strong a presumption of pus formation that immediate operation is indicated. If, by this or by other means, the presence of pus is diagnosed, operation must not be delayed. Delay for even a few hours, as, for instance, when the patient or his friends are unwilling that he should be removed to a hospital or home " until the morning," has on many occasions proved fatal from the onset of collapse of such severity that the patient's strength was not sufficient to carry him through even the shortest operation.

In addition to chronic abdominal pain, *there is a history of* CONSTIPATION, *gradually increasing to* COMPLETE STOPPAGE *of the bowels, with the gradual supervention of* VOMITING. The case is probably one of CHRONIC INTESTINAL OBSTRUCTION.

In **Chronic Intestinal Obstruction** (§ 254) the abdominal pain is more or less generalised and intermittent. The constipation may at first have alternated with diarrhœa, but after a time it is so complete that not even flatus can be passed. Vomiting, at first of food, and later of fæculent matter, a rapid pulse, and other constitutional symptoms ensue if the condition is not relieved. The four commonest causes are Malignant Stricture, Simple Stricture, Pressure of a Tumour, and Paralysis of the Lower Bowel.

The abdominal pain *is* chronic *and* GENERALISED ; *it is attended by* CONSTITUTIONAL SYMPTOMS, *and some* ABDOMINAL ENLARGEMENT *or other local signs*. The disease is probably CHRONIC PERITONITIS.

§ 260. **Chronic Peritonitis** runs a slow and chronic course, and is usually attended by a certain amount of generalised pain. There is a simple or idiopathic chronic peritonitis, but two more frequent forms are : (*a*) That due to tubercle, and (*b*) that due to cancer—two conditions which, by the way, are met with at the opposite extremes of life, and which present a very marked contrast both in their clinical and anatomical features.

CHRONIC TUBERCULOUS PERITONITIS is known by (1) the patient

is nearly always a child. (2) Pain and tenderness are present, but are not very marked features, except during subacute or acute exacerbations. (3) Fluid may be present when the disease occurs in young adults, and in acute cases, as the disease advances there is considerable tympanitic distension. Hard masses can often be felt around the umbilicus, and local tumours in some cases, the most characteristic being a sausage-shaped band produced by the rolled and infiltrated omentum running transversely across the abdomen below the edge of the liver. (4) There are emaciation and hectic fever—*i.e.*, morning temperature normal and an evening rise of 2° or 3° F., as in all active tuberculous processes. (5) Tubercle is generally found elsewhere, especially in the lungs.

Diagnosis.—The acute variety, especially when active near the cæcum, suggests appendicitis, but is recognised by the course of the disease. Cirrhosis of the liver is rare in children, and in young adults tuberculous peritonitis is not common ; both may be overlooked. In the latter jaundice and dilated umbilical veins are absent, and rectal examination may detect enlarged glands. A positive tuberculin test assists diagnosis in difficult cases. The ascitic fluid in cirrhosis contains chiefly endothelial cells ; in tuberculous peritonitis lymphocytes predominate. Inoculation of guinea-pigs with the fluid is a crucial test.

The *prognosis and treatment* are discussed in § 447.

CHRONIC CANCEROUS PERITONITIS (Cancer of the Peritoneum) is always attended by much pain, constant, and also in paroxysms. There is a great tendency to the rapid formation in the abdominal cavity of fluid which is nearly always tinged with blood. It arises only in late middle or advanced life. Its recognition is easy in typical cases on account of the age, acute pain, and ascites (under which heading it is described, (§ 209). SARCOMA of the peritoneum is rare.

CHRONIC PERITONITIS of the simple or idiopathic type is very difficult to diagnose in the majority of cases, because of the extreme variability and vagueness of the symptoms. (1) Pain and tenderness, sometimes localised, are present, worse at times and with exertion ; (2) dyspepsia, often constipation, sometimes vomiting ; (3) malaise with pyrexia from time to time ; (4) palpation may detect localised thickenings and areas of resistance ; (5) ascites is present in some cases ; in other cases it is absent, and the abdomen is very flat.

Etiology.—(1) After an attack of acute peritonitis ; (2) inflammation of any organ may cause localised peritonitis ; (3) after paracentesis without strict asepsis ; (4) idiopathic, due to unknown causes. It may occur with Bright's disease and other general conditions, in which two or more of the serous membranes (pleura, pericardium) become simultaneously affected (polyorrhomenitis or polyserositis).

The *Diagnosis* has often to be made by a process of exclusion, especially when there is no history of acute peritonitis nor of inflammation of any organ. Sometimes it is indistinguishable from tuberculous and cancerous peritonitis. Abdominal pain simulating gastralgia or colic may be due to peritoneal adhesions. When ascites reappears after repeated tappings peritonitis is usually present.

The *Prognosis* as to life is good in mild cases, though chronic invalidism is apt to ensue. Subacute attacks are liable to occur, and there may be great exhaustion and emaciation from involvement of some part of the alimentary canal, or from the formation of local abscess. Adhesions may lead to intestinal obstruction.

Treatment.—Rest and supporting belts may give relief. Inunction with blue ointment or applications of Tr. Iodine (1 in 3 of water) are useful. Paracentesis and surgical treatment may be required.

The **pain** *is of a " dragging " character,* INCREASED BY EXERTION, *accompanied by* DYSPEPTIC *and other* VAGUE SYMPTOMS. The disease is possibly DISLOCATION OF THE KIDNEY.

§ 201. **Movable Kidney** (also called Dropped, Dislocated, or Floating Kidney, according to the degree of mobility).—This condition is by no means uncommon and does not usually give rise to symptoms unless the degree of mobility is considerable.

The *Physical Signs* can only be discovered by palpation of the abdomen, with the patient lying down. The method of palpating the kidneys is given in § 318. With the patient in the erect or sitting posture, the kidney comes down more during inspiration than when lying down. After a little practice the patient will be able to lean forward and relax the muscles, which is an important aid to the observer. The left kidney rarely falls below the umbilicus, but the right one may be displaced into the iliac fossa, and even into the pelvis.

Symptoms.—In a few cases two kinds of pain may be experienced : (*a*) A constant dull, dragging pain in the back, or perhaps only an uneasiness in the loin, radiating down to the groin and inner side of the thigh, relieved by rest ; (*b*) attacks like renal colic, which may be followed by the passage of urine in large quantities, and are due to the kinking of the ureter. Such are called " Dietl's crises." Sometimes hydronephrosis results. Neurasthenia is often associated with mental depression or symptoms of dyspepsia, vertigo, diarrhœa, or constipation.

Etiology.—A very much larger percentage of women than of men have movable kidney. A fall or strain will also displace the organ, and that is why it is advisable for those with spare abdominal muscles to wear a belt when at work in the gymnasium. Attendants at refreshment bars, who have to draw beer or draw corks, often suffer from movable kidney. It is said to be extremely common among those who suffer from migraine and in those who suffer from loss of tone of other organs, as in visceroptosis (§ 203). It occurs more often in tall than in short people. Rapid loss of fat, or lowering of the intra-abdominal pressure, such as occurs after delivery, are frequent causes.

Treatment.—Bromides and rest will relieve the patient for a time, and any concurrent dyspepsia must be remedied ; but the best treatment consists in the wearing of a proper form of belt and improving the health. Operation for stitching the kidney in position is rarely needed. The abdominal belts usually supplied by instrument makers are not very successful, but an apparatus is designed for applying additional pressure outside the belt ; and in some cases pads can be introduced between the belt and the abdominal wall. Fattening of the patient is often a successful means of relieving the symptoms.

Among *the rarer causes of chronic abdominal pain may be mentioned* VISCERAL NEURALGIA (§ 197), INTESTINAL DYSPEPSIA, ENTEROPTOSIS, INCIPIENT SPINAL *or* VISCERAL DISEASE, *and* DISEASE OF THE PANCREAS.

§ 202. **Intestinal Dyspepsia** and **Intestinal Catarrh** are conditions which it is sometimes difficult to distinguish, and some doubt whether they ought to be described as separate entities. The patient complains of obscure and erratic pains in different parts of the abdomen, and of irregular attacks of diarrhœa and constipation brought on by slight dietetic errors or exercise. There is generally a good deal of flatus passed per rectum ; the fæces are offensive, very often fermenting, and contain a good deal of undigested food. When there is catarrh of the colon, there is a certain amount of mucus (see § 239) and specks of blood (not streaks, such as come from piles) in the fæces. Excess of inorganic ash in the fæces points to catarrh, especially of the colon. There may be prostration, nervousness, and ready fatigue. The disease is inconvenient

and often obstinate.   In view of the large number of organisms normally found in the intestinal contents, any continued catarrh of the intestinal wall is to be deprecated, since the bacilli make their way through a damaged wall, and thus cause what used to be called idiopathic peritonitis.   It is also probable that they enter the blood stream and give rise to coli and other infection.

Treatment.—The indications are : (1) To prevent the decomposition in the intestines ; (2) to allay the catarrh.   Recent work shows that a diet deficient in vitamins can lead to atrophy of the intestinal mucous membrane ; vitamins must be restored to the diet in such cases.   Forbid foods which are not digested—in some, fruit and vegetables ; in others, carbohydrates.   The patient may derive benefit from diet consisting entirely of milk for a time.   Constipation should never be allowed ; castor oil (which can be given in capsules) and petroleum are useful.   Intestinal antiseptics are beneficial, such as salol, dimol and lacteol.   Enemata or large antiseptic intestinal douches (3 to 8 pints) are given once a week in certain cases with excellent results. (See Colitis, § 246.)

§ 203. **Visceroptosis** (synonyms: Glénard's disease, enteroptosis, dropping of the viscera) is a condition in which there is ptosis or downward displacement of one or all of the abdominal organs.   It is ascribed to various causes, the origin of which is disputed.   Membranes are found superimposed on the lengthened mesentery.   Cases occur which have ptosis, and others which have these membranes without any symptoms.   When both conditions are present, obstructions and " kinks " tend to be formed, which give rise to varied symptoms.   Weakness of the abdominal muscles is a third factor in these cases which frequently hastens the onset of symptoms.   The *symptoms* complained of usually are : (1) Pain or dragging and " sinking " feelings in the abdomen or back, palpitation and dizziness ; (2) dyspepsia, sometimes nausea and vomiting severe enough to imitate a gastric ulcer ; (3) constipation, often alternating with diarrhœa ; (4) the intestinal stasis or delay in the passage through the intestinal canal causes symptoms of toxic absorption, such as lethargy, headache, skin pigmentation, fatigue, and nervousness.

The *diagnosis* from gastric ulcer and appendicitis in young women, and malignant disease in old people may be very difficult, even impossible ; but when palpation reveals a gurgling cæcum, and prolapse of kidneys and stomach, medical treatment can be safely tried before resorting to operation.   X-ray examination of a pronounced case shows a prolapsed stomach, the lower border reaching the pelvis, a mobile and elongated duodenum, a dilated and prolapsed cæcum, the transverse colon low in pelvis, and an elongated pelvic colon.   Lane's chief " kinks " are obstructions in the terminal coil of the ileum and of the left iliac colon due to adhesions causing pressure and narrowing of the lumen of the intestine.   X-ray examination is not always available, but the condition can usually be detected by percussion and palpation of the abdominal viscera, and on inspection in the upright position.

Treatment.—(1) The first indication is to prevent toxic absorption by aiding free elimination, and by giving food which causes less toxic residue.   The first is accomplished best by paraffin and aperients, the second by restriction of meat and abundance of fresh fruit and vegetable diet ; (2) a well-fitting and correctly applied abdominal belt such as the Curtis belt, aids mechanically by holding in better position the dropped viscera.   This gives much relief to the pain and dragging sensations ; (3) massage and electricity (rhythmic faradic or sinusoidal currents) and exercises calculated to develop the abdominal muscles, are most useful.   Rest and other measures for the relief of the nervous symptoms are also necessary.   Where all fail, operative procedure should be advised, and succeeds in certain cases.

§ 204. **Incipient or Obscure Visceral or Spinal Disease.**—(a) In cases of chronic pain GENERALISED OVER THE ABDOMEN, and in the absence of constipation, diarrhœa, or any of the causes mentioned under § 198 onwards, one might suspect cancer of the intestines, of the pancreas, or of the kidney, cancer or tubercle of the supra-renals (*i.e.*, Addison's disease, in which pain over the stomach is a constant sign), or other incipient disorders, " rheumatism " of the abdominal muscles, enteroptosis, or movable

kidney. Children may suffer from recurrent attacks of abdominal pain for which no cause can be found. Such cases should be treated as incipient intussusception—that is to say, avoid purgatives and give digestible foods and small doses of opium.

(b) In various spinal affections the pain is frequently referred to the FRONT OF THE ABDOMEN, and among the more obscure causes may be mentioned abdominal aneurysm pressing on the spine, and cancer or caries of the vertebræ. The first of these occurs mostly in male adults, the second in the aged, and the third (Pott's disease) in children. In the latter the child frequently refers to the pain as "stomach-ache," worse after running about. The girdle pain of chronic and acute myelitis should also be borne in mind.

(c) If the patient complain of PAIN SITUATED CHIEFLY IN THE LOWER ABDOMEN, one might suspect appendicitis (vide supra), cancer or other disorders of the bladder, psoas abscess, lymphatic gland enlargement, peri- and para-metritis (in which there is a good deal of pain shooting down the legs), extra-uterine pregnancy, pyosalpinx, dysmenorrhœa and all its causes, uterine neuralgia, tubercle or cancer of the prostate or testes, and obturator hernia. Hæmorrhoids are sometimes attended by pain in the abdomen (which disappears upon the cure of these), and so also are new growths and various ulcers of the lower bowel. Pneumonia or pleurisy at the base of the right lung can cause pain in the abdomen. Among the unsuspected causes I have seen pelvic hydatid in a boy of ten. The fatigue pains of debilitated women may be referred to one or other iliac region.

(d) PAIN SITUATED CHIEFLY IN THE UPPER ABDOMEN may be due to various affections of the liver, stomach, and spleen. Among the painful affections of the liver, perhaps passive congestion, gall-stones and acute cholecystitis, perihepatitis, and cancer are the commonest; hydatid is one of the obscure conditions, though it is rarely painful. Abscess above or below or within the liver should be suspected in those who have resided in tropical countries. Among the painful affections of the stomach may be mentioned gastric (or duodenal) ulcer, gastritis (acute or chronic), cancer of the stomach—which in its most usual form, scirrhus of the pylorus, is commonly very obscure in its early stages—and gastralgia. Painful affections of the spleen are not common, the chief being infarction, but the capsule is sometimes the seat of a painful inflammation. The enlargement of the organ aids the diagnosis.

§ 205. Diseases of the Pancreas are fortunately rare, for they are often unrecognisable during life. It can rarely be diagnosed with certainty, but if a combination of the following signs and tests is found, pancreatic disease is probable. On the other hand, definite disease has existed when many of the tests have been negative. When definite physical signs of tumour are present, the diagnosis is not difficult. As far as present means of investigation go, the following points are of importance in establishing the diagnosis : (1) Abdominal pain, deep-seated in the epigastrium, radiating to the left shoulder, loin, and back, with epigastric tenderness ; (2) nausea, anorexia, vomiting, diarrhœa, or constipation ; (3) debility, emaciation, and depression ; (4) creatorrhœa, or the passage of undigested muscle fibres, with striæ visible under the microscope ; (5) steatorrhœa, or the passage of liquid oil in the fæces ; (6) the passage of undigested fat in the stools ; (7) glycosuria and lowered sugar tolerance ; (8) increase of diastase in the urine and blood with decrease in the stools ; (9) Cammidge's pancreatic reaction in the urine ; (10) Sahli's test : capsules of gelatin hardened in formalin so that they are digested only by the trypsin of a healthy pancreas, and containing a drug which is readily detected in the urine ; (11) Loëwi's test : three drops 1 in 1000 adrenalin dropped on the conjunctiva and repeated five minutes later. If the pupil dilates in half to one hour, one can conclude that there is irritability of the sympathetic, which is frequent with pancreatic disease. The relationship of these signs and tests varies widely in individual cases of pancreatic disease. Severe lesions have existed without undigested fat in the stools ; steatorrhœa and creatorrhœa have existed for long without glycosuria ; Loëwi's test has been present only at intervals. Each is valuable as a link in a chain of evidence pointing to the possibility of pancreatic disease.

I. HÆMORRHAGE WITHIN THE PANCREAS, a rare condition, which, if of any extent, causes death in twenty-four hours, or less (§ 195).

II. PANCREATIC CYSTS,[1] due to obstruction or obliteration of the duct by biliary or pancreatic calculi, or cicatricial contraction. An injury to the abdomen is the chief cause. The swelling appears between the stomach and the colon, and does not move with respiration. Fatty diarrhœa is rare. The fluid withdrawn by aspiration will emulsify fat, convert starch into sugar, and digest fibrin. The prognosis is good with surgical treatment.

III. PANCREATIC CALCULI are small concretions consisting chiefly of carbonate of lime. They are visible on X-ray examination, a diagnostic feature which distinguishes them from the majority of biliary calculi.

IV. ACUTE PANCREATITIS is met with in three forms : (1) *Acute Hæmorrhagic Pancreatitis*, which sets in suddenly with agonising pain, and results in death in one to four days (§ 195). (2) *Acute Suppurative Pancreatitis* begins suddenly with pain and irregular pyrexia, and may lead to death in three or four hours, but Fitz's cases more often became chronic, and lasted some months. There may be several small or one large abscess. (3) *Gangrenous Pancreatitis*, in which necrosis of the organ occurs, and it may be passed as a slough by the bowel. Two of Fitz's cases recovered.

V. CHRONIC PANCREATITIS is a fibrosis of the organ which mostly runs a latent course, because it is frequently associated with diabetes, especially in those cases where atrophy of the gland ensues. The onset is insidious ; discomfort and distension in the epigastrium are felt after meals, and drowsiness. Borborygmi and offensive stools, anæmia, and emaciation follow. Paroxysmal pain is complained of above and to the right of the umbilicus, and tenderness can be elicited there. The pain may be referred to the left scapula. Later, the infective process invades the bile ducts and produces jaundice, with dilatation of the gall-bladder, and thus resembles gall-stones and cancer of the head of the pancreas. Later still, there may be pressure on the duodenum and vena cava.

The diagnosis is difficult in early stages, and requires expert analysis of the excreta. Cammidge's " pancreatic reaction " and the presence in the urine of indican, calcium oxalate crystals, bile, and urobilin, are suggestive of pancreatitis. Later, the stools are fatty and so characteristic that the condition can be diagnosed by the naked eye.

PANCREATIC DIABETES.—The association of glycosuria with pancreatic calculus was first pointed out by Cowley in 1788. But it was Lancereaux, in 1877, who maintained there was a special form of diabetes dependent on grave alterations in the pancreas (Pancreatic Diabetes), characterised by poluria, excessive thirst and appetite, rapid loss of flesh, and glycosuria. Pancreatic diabetes may, however, occur with lesions of the pancreas other than chronic pancreatitis ; and grave alterations of the organ may exist without diabetes.

VI. CANCER OF THE PANCREAS may be primary or secondary, and is a rare condition. It is said to occur in about 6 per cent. of all cancers (Segre).[6] The symptoms are : (1) Pain in the epigastrium, which at first occurs in paroxysms, then becomes constant, and runs a chronic course. (2) Symptoms of gastric disorder may be present for months before any other symptom. (3) Jaundice, intense and persistent from the pressure on the bile-duct, is usually present, and sometimes pain like biliary colic accompanies this. (4) The other symptoms are those above described. (5) Later on a tumour is found in the epigastrium or in the umbilical region, with little or no mobility, deep-seated, and hard to define. (6) Œdema of the legs, from pressure on the inferior vena cava, may occur.

The *Diagnosis* of cancer and other tumours of the pancreas is difficult. A tumour

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[1] A case of retroperitoneal rupture of a pancreatic cyst occurring in a young man about twenty-five years of age was admitted to the Paddington Infirmary with all the symptoms of acute peritonitis. Laparotomy was performed by Sir Frederick Treves, but nothing was found until after death, forty-eight hours later. The origin of the cyst was not even then discovered, but the cellular tissue behind the peritoneum was infiltrated with the usual pultaceous material.

of the liver, pylorus, or transverse colon, is more mobile ; pancreatic tumours do not move with respiration. Much indicanuria points to an intestinal rather than to a pancreatic tumour. No great stress can be laid on the presence of fat in the fæces, or on glycosuria, but abundant undigested muscle fibre found in the fæces is more characteristic of pancreatic disease. Increase of diastase in the urine and a positive result with Loëwi's and Cammidge's tests point to pancreatic disease.

Prognosis.—In cancer of the pancreas death usually occurs soon after the onset of jaundice, or six weeks after ascites sets in. Emaciation and debility may not come on till late in the disease. The complications are : (i.) Symptoms due to pressure on the neighbouring organs—intestine, stomach, or portal vein ; (ii.) sudden hæmorrhage into the alimentary tract or the peritoneal cavity ; (iii.) pulmonary embolism. Sudden death occurs in the last two.

Treatment is mainly symptomatic. Starches and sugars should be limited. Milk and casein are the most digestible forms of proteid in pancreatic disease. The administration of pancreatin, pancreon, or similar preparations may aid the digestion. Duodenal catarrh may be allayed by bismuth salicylate ; and urotropin disinfects the biliary passages. Opening and draining the gall-bladder has been successful in cases of pancreatitis accompanied by jaundice, and other surgical measures are employed for the several diseases of the pancreas.

GENERALISED ABDOMINAL ENLARGEMENT

Difficulty in the diagnosis of the cause of abdominal enlargement can often arise in cases of pregnancy, venous congestion, atony or ptosis of the abdominal organs. And see Fallacies, § 190, for the less common sources of error.

§ 206. Classification.—Generalised abdominal enlargement occurs under four conditions :

The **Routine Procedure,** as previously described (§ 191), should be by Inspection, Palpation, Percussion, Auscultation, and Mensuration.

If a **hard tumour** can be felt in any part, turn first to § 212.

If the **abdomen** is quite **soft to palpation** and **resonant** all over, turn first to § 207.

If the abdomen is **dull to percussion** in the flanks, and presents the fluctuation test, turn first to § 209.

If the abdomen is **resonant in the flanks** and **dull in front,** turn first to § 210.

The abdomen is **uniformly enlarged** *; it is* SOFT *and yielding to palpation ; and percussion, systematically conducted over the whole area, gives a* RESONANT *note.* The swelling is probably due to TYMPANITES.

§ 207. Tympanites is the term employed for a flatulent distension of the stomach and intestines by gas. It should be remembered that flatulent distension may accompany and render obscure a *small quantity of fluid in the peritoneum.*

The *Causes* of tympanitic enlargement are as follows :

I. Atonic and other forms of DYSPEPSIA are the most frequent causes of flatulent abdominal distension. It is usually intermittent, and generally greatest after meals (§ 228). Acrophagy, or air swallowing, is a common cause.

II. In ATONY OF THE COLON the bowels are constipated, and the patient is liable to "colicky" pains ; and the constitutional symptoms are few except when there has been prolonged toxæmia (§ 252).

III. In TUBERCULOUS PERITONITIS there is a tendency to the formation of intestinal adhesions and *flatulent distension*. In tuberculous peritonitis, moreover, the distended abdomen has a doughy feel and here and there a patch of dulness on percussion, which is quite characteristic (§ 200).

IV. "PHANTOM TUMOUR" may assume the shape of a generalised more or less resonant enlargement, but it more often resembles a localised tumour (§ 212).

V. In OBSTRUCTION OF THE BOWELS there is considerable abdominal distension, accompanied by pain, vomiting, and other general constitutional disturbance (§§ 253 and 254).

Gas in the Peritoneal Cavity gives much the same signs as tympanites, only there is extreme distension, and hyper-resonance all over to such a degree that the normal dulness of the liver and spleen is obscured. It is met with only when perforation of some part of the alimentary canal occurs. The patient is collapsed, and presents all the symptoms associated with perforation (§ 193). A few hours after the occurrence of the perforation a delusive lull occurs in the collapse and other symptoms, only to be succeeded by a fatal exacerbation. Perforation of gastric ulcer is the commonest cause, and one of the diagnostic features of this condition is the loss of the normal area of liver dulness.

There is uniform abdominal enlargement, *which is soft and yielding to palpation and* DULL TO PERCUSSION *in parts ;* the FLUCTUATION SIGN *is present*. There is FLUID WITHIN THE ABDOMEN.

§ 208. When there is **Fluid in the Peritoneal Cavity,** either free or encysted, the belly is soft to palpation, dull to percussion in parts (either in the flanks or in front), and the measurements show the abdomen to be uniformly enlarged.

When the fluid is in any quantity, two special signs can be elicited. (1) *Fluctuation test*.—When a large amount of fluid is present, a wave of *fluctuation* may be *seen* to travel *across the surface* when we tap or "flip" one side. This can only be satisfactorily elicited when the abdomen is full and tense. (2) *Percussion test*.—A percussion wave can be transmitted from one hand to the other *through the fluid* by the law that fluids transmit pressure or a blow equally in all directions. Place the left hand over one side of the dull portion, and tap sharply with the fingers of the right hand over the opposite side : an impulse will be felt by the left hand if fluid be present. In applying the "percussion test"

for fluid, an assistant should place the edge of his hand vertically on the umbilicus. This will prevent the wave or impulse from travelling across the surface of the omental and subcutaneous fat instead of through the fluid. Neither of these signs can be elicited in a gaseous enlargement or a solid tumour. In *obese persons* considerable difficulty arises in the detection of fluid.

The fluid may be either (a) FREE in the peritoneal cavity, when it is termed ascites ; or (b) enclosed in a CYST, such, for instance, as an ovarian cyst.

(a) If FREE in the peritoneal cavity, it will obey the law of all fluids, and *shift with the position of the patient.* Thus in ascites (§ 209) when the patient is on his back you will find both flanks are dull to percussion, and the umbilical region is resonant ; then, if the patient turns on one side you will find that the uppermost flank which before was dull is now resonant, while the umbilical region, if there is much fluid, is dull. Much may be learned from the character of the fluid withdrawn by a trocar. Ascitic fluid is straw-coloured, with much albumen. Hæmorrhagic fluid usually means tubercle or cancer.

(b) If the fluid is ENCYSTED—*e.g.*, ovarian cyst, we can still elicit the fluctuation and the percussion tests just referred to, but the level of the dulness will not alter with the position of the patient (§ 210).

There is a generalised uniform enlargement of the abdomen, which gives all the SIGNS OF FLUID, *and the fluid* ALTERS ITS LEVEL *with the position of the patient. The condition is* ASCITES.

§ 209. Ascites is a term applied to an effusion of non-inflammatory fluid within the peritoneum (dropsy of the peritoneum). The physical signs of fluid have just now been described above. It is sometimes difficult to detect a very small quantity of fluid in the peritoneum, but its existence is rendered probable (i.) by the dulness on percussion of the umbilical region with the patient on his hands and knees, and (ii.) by finding that when the patient turns from one side to the other, the flank which was dull is now resonant.

Ascites may have to be *Diagnosed* from any of the cystic conditions mentioned below (§ 210), but certainly the most frequent and important source of difficulty is *ovarian cyst.* In ascites (i.) the flanks bulge, (ii.) the front is flat and resonant, and (iii.) both flanks are dull, but if the patient turns on his side the upper flank becomes resonant—three features which are the exact reverse of those found with ovarian and other cystic tumours (see also table on p. 287). Occasionally peritoneal adhesions (especially cancerous) may confine the fluid to one part of the abdomen, and then the fluid does not shift with the position of the patient. A greatly distended urinary bladder may simulate ascites, but the passage of a catheter readily excludes this fallacy.

The other *Symptoms* which accompany ascites belong to two categories : (1) Those due to pressure within the abdomen—*e.g.*, œdema of the feet

and legs, from pressure on the vena cava and its branches; later on dilatation of the surface veins of the anterior abdominal wall may occur from the same cause; albuminuria from pressure on the renal veins, and dyspnœa from mechanical impediment in the lungs or in the circulation. (2) There are evidences of the condition which has caused the ascites, and of all the causes by far the commonest is alcoholic cirrhosis of the liver. The temperature is generally normal, except in chronic peritonitis.

The *Causes of Ascites* are five in number. In reference to the diagnosis, of these causes, if there be any œdema of the ankles, it is important to ascertain whether this œdema or the ascites came first. For instance, when PORTAL OBSTRUCTION is in operation, the dropsy of the feet will have started subsequently to the ascites; in HEART OR LUNG disease it will have preceded the ascites; whereas in RENAL DISEASE they would have started about the same time. ASCITES with well-marked JAUNDICE in an old person is extremely likely to mean CANCER OF THE LIVER or peritoneum ASCITES with SALLOWNESS of the skin in a MIDDLE-AGED person is most probable due to ALCOHOLIC CIRRHOSIS of the liver.

I. **Portal Obstruction** is the commonest cause of well-marked ascites. This is recognised in two ways : (*a*) By a history or presence of the *symptoms* of portal obstruction (of which ascites is only one); and (*b*) the presence or a history of one of the *causes* of portal obstruction.

(*a*) The SYMPTOMS of portal obstruction, in the order in which they usually appear, are as follows : (1) A liability to attacks of gastric and intestinal catarrh, as evidenced by pain in the stomach, irritable dyspepsia, alternating diarrhœa and constipation, and the vomiting of mucus streaked with blood, especially in the early morning before breakfast. (2) Hæmorrhoids and enlarged œsophageal veins. (3) Hæmorrhage, sometimes in very large quantity, from the stomach and the bowels. (4) Congestion, and therefore enlargement of the spleen. (5) ASCITES is one of the later results. (6) Enlargement of the veins of the abdominal wall from the establishment of a collateral circulation. (7) Œdema of the legs also appears subsequent to the ascites, and is due to pressure on the large viens in the abdominal cavity by the ascitic fluid. (8) Albumen in the urine may arise in the same way, or from concurrent disease of the kidney ; in the former case the albuminuria may disappear after paracentesis.

(*b*) The CAUSES of portal obstruction may be grouped into (*a*) diseases within the liver, or (*β*) diseases outside it.

(*a*) *Diseases within the Liver.*—*Cirrhosis* of the liver is by far the commonest of all the causes, and this is nearly always due to alcoholism, there being a history of this and of alcoholic dyspepsia. Simple ascites without marked jaundice or other obvious symptoms is presumptive of cirrhosis. *Cancer* produces portal obstruction usually by the pressure of the enlarged glands in the fissure, or by masses protruding outside the liver. *Perihepatitis* sometimes produces ascites by puckering of the capsule. Ascites only very rarely accompanies *hepatic congestion*, and, never fatty liver, hydatid, or abscess.

(β) The causes of portal obstruction *outside the liver* are : (1) *Cancer* of the stomach, duodenum, or pancreas, and various other tumours pressing on the vein.   (2) Enlargement of the *glands* in the fissure of the liver (cancer, tubercle, syphilis or lymphadenoma).   (3) *Thrombosis* of the portal vein is rare, and the symptoms are very acute.

II. In **Heart Disease**, either primary (*i.g.*, mitral disease and cardiac dilatation) or secondary to lung mischief, the ascites is generally part of the dropsy affecting the cellular tissues and other serous cavities of the body.   Here dropsy of the *feet will have preceded the abdominal dropsy*, and there will be a previous history of palpitation, dyspnœa, and perhaps cough.   An examination of the heart will also reveal the nature of the disease.

III. In **Kidney Disease** ascites may be part of a General Dropsy affecting the face, limbs, peritoneum, pleuræ, and pericardium.   The fact that the dropsy started in all of these situations about the same time reveals this cause.   Albuminuria is frequently enough a consequence of the pressure of the ascitic fluid, but the presence of epithelial casts almost certainly indicates that the renal disease was primary.   It usually takes the form of acute or chronic parenchymatous nephritis, rarely waxy or granular kidney.

IV. **Chronic Peritonitis** is another cause of fluid in the peritoneum. An idiopathic form of chronic peritonitis is sometimes described, but it is practically never met with apart from a deposit of tubercle (in the YOUNG) or of cancer (in the AGED), § 200.   In the tuberculous form adhesions rather than fluid are met with ; in the cancerous it is *vice versa*.

V. A small amount of effusion into the peritoneum is found in severe **anaemia** and some other blood disorders ; but it is never very great.

VI. Chylous ascites, or the collection of chyle in the peritoneal cavity, occurs as the result of obstruction of the thoracic duct, or it may occur after trauma, or in spleno-medullary leukæmia.   In tropical countries it is more often due to the adult *Filaria sanguinis hominis*.

The *Prognosis and Treatment of Ascites* are very largely those of the morbid condition with which it is casually related.   The *Prognosis of Ascites due to portal obstruction* depends very much on the nature of the intra- or extra-hepatic lesion which has produced it, as given above and in Chapter XII.   The degree of the obstruction is measured by the amount of ascites and other symptoms present, and still better by the amount and frequency of the hæmorrhage that has taken place from the stomach or intestines.   Life may be prolonged for many years even when a considerable amount of ascites has accrued, provided it has come on slowly, and time has thus been afforded for the gradual establishment of the collateral circulation through the surface veins of the abdomen and other collateral channels.   It is in this sense that repeated tappings are good, for in this way time is gained for the establishment of collateral circulation. In cases of alcoholic cirrhosis the habit must be abandoned, otherwise

the patient cannot live longer than six to twelve months, for ascites indicates an advanced condition of cirrhosis; in cases treated early, recovery may be complete.

The *Treatment of Ascites*, like its prognosis, must depend upon its cause (*q.v.*). The treatment of *ascites due to portal obstruction*, and to some extent that of other forms, is as follows: (1) Hydragogue purgatives are certainly called for, and mag. sulph. and the other salines are the best. Elaterium seems particularly valuable if given in sufficient quantities to produce three or four watery stools a day. (1) Diuretics are recommended by some, but in my experience there is no form of dropsy in which they are of so little use as in ascites, at any rate until the pressure has been relieved by tapping. Diuretin, copaiba resin, and cubebs are useful, and I have given pil. digitalis co. with some benefit after repeated tapping. (3) Tonics are useful combined with the preceding, such as a mixture containing pot. bitar., fer. tart., and digitalis. (4) Paracentesis is generally called for sooner or later. Some physicians say it should be put off until it is called for by the urgency of dyspnœa. In cancer this is certainly a good rule, but in cirrhosis of the liver it is best to operate at once in all cases where there is much fluid, unrelieved by medicine. It is often found that medicines which were useless before are efficacious after the operation, because the kidneys are relieved from pressure. Sometimes complete recovery takes place after repeated paracentesis, because time is thus afforded for the establishment of the collateral circulation as above mentioned. It is best to use a small trocar with the tube conducted to a pail, so that the peritoneum may gradually empty itself. With a large one leakage may remain, or peritonitis may ensue. In 1896 the Talma-Morison surgical method of promoting the collateral circulation by the artificial production of omental adhesions in cases of alcoholic cirrhosis was introduced, and has been attended by a measure of success.

There is a generalised abdominal enlargement which gives all the SIGNS OF FLUID (§ 208); *but the fluid does* NOT ALTER ITS LEVEL *with the position of the patient.* There is ENCYSTED FLUID (probably ovarian) IN THE ABDOMEN.

By far the commonest of such cystic tumours is an OVARIAN CYST. Other and less common cystic abdominal tumours are HYDRAMNIOS, CYSTIC FIBROMA of the uterus, HYDRO- and PYO-NEPHROSIS, PANCREATIC CYST, a large HYDATID, a cyst of the GALL-BLADDER, and an ENCYSTED ASCITES.

§ 210. I. Ovarian Cyst is centrally situated, and grows from below upwards. The enlargement is fairly uniform, and it gives all the signs of fluid (§ 208). But the level does not alter with the position of the patient; and whereas the umbilical region is dull on percussion, the flanks are resonant. On palpation it is tense and elastic, and in malignant ovarian cysts nodules can be felt in the walls. The diagnostic features between ascites and ovarian cysts are given in Table XIV.

TABLE XIV.

	Ascites.	*Ovarian Cyst.*
Inspection.	Flanks bulge, front flat.[1]	Flanks flat, front bulges.
Percussion.	Flanks dull, front resonant. On turning, upper flank becomes resonant.	Flanks resonant, front dull. No alteration of dulness on turning.
Measurement.	Umbilicus to xiphoid greater than umbilicus to pubes. Circumference at umbilicus greater than slightly below. Navel to iliac spine same both sides.	Umbilicus to xiphoid less than umbilicus to pubes. Circumference at umbilicus less than slightly below. Navel to iliac spine greater one side than another.

The features associated with it are (1) a history of it having grown upwards from the pelvis, and (2) these tumours (unlike encysted ascites) may be of very rapid growth, and reach quite a large size in three or four months. (3) There have usually been menstrual irregularities, though by no means always. There may have been no general symptoms of any kind, but generally some pain and local discomfort have been complained of. Often when the cyst contains pus there is little or no fever. When there is a history of attacks of pain, it generally indicates adhesions, an important matter from an operator's point of view. An examination of the uterus usually reveals nothing. A malignant cystic ovarian growth is indicated by (1) the presence of nodules in the walls; and (2) the age of the patient and a history of emaciation, and severe pain.

Diagnosis.—In the *earlier stages* the diagnosis of an ovarian tumour is sometimes difficult. It is an elastic, movable, and globular swelling; the uterus is not enlarged, and it can be defined as quite separate from the tumour. In this stage it may have to be diagnosed from *hydro-* or *pyo-salpinx.* *Para-* and *peri-metric* exudation and *pelvic hæmatocele* would be very firmly fixed in the pelvic cavity and accompanied by constitutional symptoms. In *extra-uterine fœtation* there would be morning sickness, a patulous os uteri, and other symptoms of pregnancy, with an empty uterus.

In the *later stages* ovarian cysts have to be diagnosed from all the conditions mentioned below.

II. PREGNANCY WITH HYDRAMNIOS and a thin uterine wall is sometimes very difficult to diagnose from an ovarian cyst, for both develop very rapidly. Experienced clinicians have been known to fail in the differentiation. The symptoms of pregnancy (see § 355), the exactly central position of the tumour, and the softened cervix, may aid us in the diagnosis. The serum test for pregnancy should be tried. *Hydatid mole* presents similar difficulties, but it is fortunately more rare.

III. LARGE CYSTIC FIBROID of the uterus, especially of the subperitoneal variety, may produce the signs of a fluid tumour. It is recognised by (1) its connection with the uterus, which is enlarged; and (2) its slow growth, which may extend over many years; and (3) menorrhagia in some cases.

IV. A LARGE HYDATID CYST of the spleen or liver, a HYDRO- or PYO-NEPHROSIS,

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[1] Bulging in front may occur in cases with large and acute effusion.

a dilated GALL-BLADDER, a large PANCREATIC, OMENTAL, or MESENTERIC CYST, or a large PERITYPHLITIC ABSCESS, may on rare occasions produce the appearance of a general fluid enlargement of the abdomen, and may require to be diagnosed from ovarian cyst; but they are nearly always *asymmetrical.* They grow from, and their percussion dulness is continuous with, the organs whence they rise; they are referred to among Abdominal Tumours (§ 211).

V. ENCYSTED ASCITES is not common. It may result from previous peritonitis, of which there will probably be a history. More frequently, perhaps, it results from tubercle or cancer of the peritoneum (§ 200). In very rare cases congenital deficiency or adhesions may exist. In all of these there is a want of symmetry in the enlargement and in the fluid, an absence of the associated symptoms of ovarian tumour, and a history or other evidences of the cause in operation.

The *Prognosis* of ovarian tumour is always serious, though in the non-malignant form it may be quiescent for some years. If not treated, a cyst may go on (1) to rupture and fatal peritonitis; (2) it may become inflamed; (3) the pedicle may become twisted; (4) hæmorrhage may take place into its cavity.

The *Treatment* is entirely surgical. The earlier the cyst is removed the better. It is best to do this before the occurrence of attacks of pain indicating inflammatory adhesions. Tapping is a temporary measure only; it certainly increases the risks for future operation, and is justifiable only in elderly patients where operation is for some reason impossible.

ABDOMINAL TUMOURS.

§ 211. **Method of Procedure.**—We now turn to the second group of abdominal enlargements—namely, those in which the enlargement has originated in, or is localised to, one part—*i.e.,* Abdominal Tumours. It is only by repeated and careful examination that mistakes can be avoided in the diagnosis of abdominal tumours. The same methods are adopted here as in general enlargement (§ 191), which should be consulted. (1) *Inspection* in the recumbent, and sometimes in the erect, posture *should never be omitted;* (2) *Palpation,* with a flat hand previously warmed and with the patient's abdominal muscles thoroughly relaxed by a suitable posture; (3) *Percussion,* to define the boundaries and nature of the tumour, and its continuity with some organ; (4) careful *Measurement* made and recorded both for the comparison of one part with another, and to note the progress made by the growth; and (5) *Auscultation,* which is especially useful in the diagnosis of late pregnancy.

Fallacies of Abdominal Tumours.—(1) *Obesity* may offer a serious obstacle to the examination of abdominal enlargements or tumours. In these cases the umbilicus is usually depressed. The only way to arrive approximately at a correct decision is to place the hand flat upon the belly and then dip the fingers suddenly and forcibly inwards.

(2) The presence of *fluid* within the abdomen, together with a solid tumour, may prevent our discovering or examining the latter thoroughly. The difficulty may be obviated to some extent by suddenly flexing the fingers as in the case of obesity.

(3) *Fœcal accumulations* may simulate malignant and other tumours, though they can generally be indented by the fingers. They are always situated in some part of the large bowel. In doubtful circumstances a course of castor oil or other hydragogue purgative is desirable. But they may exist for many weeks in spite of purgatives.

(4) A "*phantom tumour*" is a swelling (usually tympanitic, sometimes dull), produced by irregular muscular contraction, and it is wonderful how precisely it may simulate a solid tumour. It is apt to appear and disappear suddenly, hence the name. The condition is met with for the most part in young hysterical women, and is usually beyond the control of the patient. It is a frequent cause of error in diagnosis. It is generally due to spasmodic contraction of one or both recti muscles. Spasm of the diaphragm may produce a generalised abdominal enlargement by pushing the viscera down. The patient should be placed in a position of perfect ease for the relaxation of all the muscles of the body, with the knees drawn up and the neck slightly bent. Sometimes nothing but the administration of an anæsthetic to complete narcosis will enable us to establish the diagnosis, and this must be done in cases of importance.

(5) The liver occasionally presents the abnormality of an extra lobe. Displaced or movable organs may be mistaken for tumours. (See § 190.)

Having excluded these fallacies, and being satisfied as to the existence of an abdominal tumour, there are five points to which our attention should be directed :

1. The first and most important question is the *locality of the tumour*, in which region is it situated, or where did it start ?

2. To ascertain with *which organ it is connected*, consider what organs are located in the region occupied by the tumour, and then see if it be structurally continuous by palpation and percussion with one of these.

3. If it *moves with the breathing* of the patient we know that it must be connected with the diaphragm, or some organ depressed by it during respiration, such as the spleen, liver, stomach, intestines, or omentum. If fixed, it is a tumour of the kidney (unless it be dislocated), pancreas, aorta, lymphatic glands, or some other organ unaffected by respiration, or bound down by adhesions.

4. Inquire for a *history of any disease* or functional disturbance of the abdominal organs—*e.g.*, in the case of the kidney, whether the urine contains, or has contained, blood or pus—although the tumour may appear to be far from these organs ; or perhaps there has been jaundice pointing to hepatic mischief.

5. The diagnosis of the *nature of the tumour* depends very largely upon its history and the age and sex of the patient. Tense cystic tumours are extremely difficult to differentiate from solid growths, but we can try to obtain the percussion and fluctuation tests (§ 208). There is also another question which very frequently presents itself for consideration —viz., is the tumour benign or malignant ? The general symptoms of malignant disease (cancer) are discussed in § 445 ; but the age of the

patient, and the rapid course and lethal tendencies of the disease, are the chief means of differentiating it.

§ 212. *If there is a visible or palpable* **tumour,** *in the abdomen, ascertain which* REGION *the tumour chiefly* OCCUPIES *or* ORIGINATED *in, and refer to that region in the following summary. Having identified* ITS ORIGIN *in this way, reference must be made to the diseases of the organ affected to ascertain the* NATURE *of the tumour.*

I. RIGHT HYPOCHONDRIUM.—The commonest tumours in this position are tumours of the *liver,* especially cancer and enlargement of the organ. The features which HEPATIC TUMOURS present in common, in addition to their position, are : (1) They are not covered in front by resonant bowel, and their dulness is continuous with that of the liver ; (2) they move with respiration ; and (3) there are ascites, jaundice, and other evidences of liver derangement. It must not be forgotten that hepatic tumours may be simulated when the liver is pushed down by emphysema, or by pleuritic and pericardial effusions ; or that it may be puckered by contraction of the capsule, and so simulate a tumour or enlargement (Diagnosis of Hepatic Enlargements, § 260) ; Riedel's lobe (see below) is another fallacy. A distended GALL-BLADDER (*e.g.,* by gall-stones) is recognisable as a tense rounded swelling below the ninth costal cartilage. There is only occasionally a history of biliary colic but always a history of " chills " (biliary fever), see § 266. It is distinguished from the kidney by the fact that the colon passes over the kidney. Tumours in this region may also be connected with the *duodenum* or *right kidney* (see II. and IV.).

Riedel's Lobe (lingiform or floating lobe of the liver).—In certain cases, sometimes associated with gall stones retained within the gall-bladder, a tongue-shaped process projects downwards from the right lobe of the liver, or the lobus quadratus. It may reach as far as the iliac crest, or even to the iliao fossa. In hardly any of the cases in which it has been observed (Glénard collected eighty), has the condition been correctly diagnosed until operation or an autopsy was performed. It has most often been mistaken for floating kidney, and has also been taken for distended gall-gladder, hydatid cyst, new growth, and omental tumour. It is sometimes tender, its shape more or less that of a pear. Under chloroform its connection with the liver might possibly be made out.

Suprarenal Tumours become manifest in the right or left hypochondrium, and are difficult to distinguish from tumours of the liver, gall-bladder, kidney and spleen respectively. Mayo Robson summarises the symptoms thus : (i.) Pain radiating from the tumour across the abdomen and to the back ; (ii.) pain complained of at the shoulder tip ; (iii.) emaciation, with nervous depression, and digestive disturbance ; (iv.) a tumour felt beneath the costal margin (right or left), at first movable with respiration, but soon fixed ; and it can be felt posteriorly in the costo-vertebral angle ; (v.) absence of urinary and gall-bladder symptoms. More recently other symptoms have been described in relation to suprarenal tumours. In male children precocious general and sexual development may occur, and sometimes strong muscular development. In female children there may be amenorrhœa, defective mammary glands, hirsuties, obesity and a deep voice. When the tumour is sarcomatous, there is a special tendency to secondary affection of the bones, particularly those of the skull, and to exophthalmos, which may occur before any abdominal tumour can be felt. Dr. R. S. Frew finds a different syndrome of symptoms according to whether the

primary sarcomatous growth affects the right or the left suprarenal. When the left
is involved, exophthalmos appears first on the left side, and pain in the limbs is
common.

II. In the EPIGASTRIC REGION tumours may be connected with the
liver (*vide supra*) ; but the first tumour which would occur to one's mind
would be CANCER OF THE STOMACH—*i.e.*, a hard, irregular swelling attended
by vomiting, "coffee-ground" in character. The commonest form of
•malignant disease of the stomach, however, is scirrhus of the pylorus, in
which condition copious vomiting at long intervals and other gastric
symptoms appear long before any swelling can be detected (§ 232).
Tumours of the duodenum may sometimes be distinguished from those of
the stomach by their immobility during a deep respiration.

Pancreatic cysts may cause a fluctuating swelling in the epigastrium, but their
detection is extremely difficult. There may be a history of pain, and symptoms of
pancreatic disease (see § 205). Cysts of the *small omental sac* present a similar swelling.
Pulsation in the epigastrium may be due to dilated right ventricle but is usually normal ;
rarely it is caused by abdominal aneurysm.

III. In the LEFT HYPOCHONDRIUM tumours of the SPLEEN originate,
and sometimes they attain to an enormous size. These are fully discussed
in § 286. They move with respiration, and they make their way forward
in *front* of the colon. A splenic tumour can generally be moved forwards
by getting the hand behind it, a fact which distinguishes it from tumour
of the left kidney, and it presents the characteristic splenic notch (§ 286).
It resembles tumour of the left lobe of the liver, but the latter cannot be
displaced downwards by the hand. Other tumours in this position may
be connected with the *stomach, pancreas, liver, kidney,* and *sigmoid flexure.*

IV. The LUMBAR REGION may be the starting place for RENAL
TUMOURS, which are characterised by four features : (i.) Their fixity
during respiration. (ii.) Dulness in one flank, and, unless, both kidneys
are involved, resonance in the other. (iii.) They are *always resonant in
front,* because as they make their way forward they push the colon in
front of them ; and (iv.) there is no resonant part between the dulness
of a renal tumour and the spine, as there would be in the case of a splenic
tumour. In many the rounded and reniform shape of the kidney is
retained. They are distinguished from hepatic tumours by the dulness
in the flank not being continuous with that of the liver, and by the presence
or history of blood, pus, or other urinary changes. The commoner forms
of renal tumours are hydro- and pyonephrosis, renal sarcoma (commonest
tumour in children), and perinephric abscess. A perinephric abscess tends
to bulge backwards. *Pyo-* or *Hydronephrosis* are cystic tumours, con-
taining urine *with* or *without* pus respectively (see § 341). Hydronephrosis
may be almost painless, not tender, and unattended by any subjective
or constitutional symptoms ; pyonephrosis is always tender, and attended
by hectic fever and malaise. Hydatid of the kidney may only be evidenced
by swelling ; sometimes it gives a thrill on percussion. Other tumours

starting in the lumbar regions may be connected with the *ascending* and *descending colon.*

Movable or *Floating Kidney* is one of the most frequent of abdominal tumours. It may be found in any part of the cavity below the liver. Its mobility, rounded or reniform shape are characteristic, but not always easily detected. There is a characteristic pain of a dull, aching, or dragging character in the back, increased by exertion (see § 201).

V. The LEFT ILIAC REGION may be the seat of a tumour caused by CANCER of the SIGMOID FLEXURE, and this is the most frequent position in the bowel for cancerous growth. Cancer and other *tumours of the large intestines* are distinguished generally by their free mobility (unless fixed by adhesions). They are, when cancerous (far the commonest neoplasm of the intestines), attended by irregularity of the bowels, generally chronic diarrhœa. The commonest starting-point for primary cancer of the bowel is, as just mentioned, the sigmoid flexure ; but before a cancerous swelling can be detected in the left iliac region the patient will have been troubled with recurrent diarrhœa and pain, sometimes melæna. These symptoms are followed in course of time by œdema of the leg or sciatica. In *cancer* of the peritoneum all the intestines may become matted together, and although fluctuation may be detected, there is little or no fluid in the peritoneal cavity. Sarcoma of the *small intestines* gives rise to hard, irregular, nodular, usually multiple tumours, and, in addition to the signs just mentioned, there are the advanced age of the patient and cachexia. Constipation, going on sometimes to obstruction, may also be present. The prognosis of cancer is given in Chapter XVI. But so-called " colloid cancer " of the peritoneum is a remarkable exception in regard to its duration, and it may go on for years before death occurs. The treatment, which is not very hopeful, is referred to under " Emaciation."

Diverticulitis.—While diverticulitis or hernia and dilatation may occur in any part of the alimentary canal, the condition known as diverticulitis signifies inflammation of a sacculated portion of the sigmoid. It occurs in males, rarely before the age of sixty. There is a history of chronic pain in the left iliac region, and palpation reveals a swelling, the nature of which is made evident on expert X-ray examination. Pyrexia occurs from time to time. It may be mistaken for cancer, from which it is distinguished by the absence of blood in the stools, and the length of the history without any general emaciation. The disease may become acute, with perforation or gangrene, or there may be recurrent attacks of inflammation. Occasionally there is spontaneous recovery.

VI. The RIGHT ILIAC REGION is the position in which APPENDICITIS is usually manifested ; it is fully described under " Abdominal Pain " (§ 199). *Intussusception* of the bowel, which occurs mostly in childhood, gives rise to a soft, sausage-shaped swelling generally situated in this region (§ 253). *Pelvic cellulitis* may form a firm swelling in either iliac region. Its other features are (i.) vaginal examination reveals a tender swelling in the corresponding fornix, pushing the uterus to the opposite side ; (ii.) there is a history of acute pain and fever at the onset of the condition, frequently following childbirth or abortion. Tubercle and

cancer of the cœcum, contrary to what we might expect, often constitutes a *movable* tumour in the iliac region, and is very apt to be mistaken for masses of fæces. Cancer of the cæcum may be attended by suppuration, so giving rise to abscess in this region with pyrexia. The history of such cases may run a long course, and, except in the age of the patient, resemble chronic appendicitis. Enlarged glands in this region have often been mistaken for chronic appendicitis. *Psoas abscess* may point in this region. A right movable kidney may simulate a tumour in this region.

VII. The UMBILICAL REGION is the starting place of tumours connected with the pancreas, duodenum, mesenteric glands, and aorta, all of which are *immobile during respiration;* though a tumour in this position is far more often connected with the stomach, liver, or large bowel, which *move with respiration.* Enlargement of the *mesenteric glands* may be sometimes detected in spare subjects by grasping the two sides of the abdomen either between the two hands or the finger and thumb of one hand. When large enough to form a tumour, they are fixed and matted together.

Aneurysm of the Abdominal Aorta is a pulsatile and expansile swelling also immobile during respiration. In thin subjects a thrill may be felt, and a murmur heard. In auscultating the abdominal aorta we must be careful not to produce a murmur by pressure of the stethoscope. It is attended always by a severe fixed neuralgic pain in the spine, and sooner or later breathlessness and cardiac signs. It is these latter symptoms which distinguish true aneurysm from " pulsatile aorta " (see below), and from a swelling in front of the vessel to which the pulsation has been communicated. An endeavour should be made to grasp the swelling on each side, so as to observe the expansile nature of the tumour.

Pulsating Abdominal Aorta (throbbing in the belly).—Dyspeptic subjects and nervous females are often troubled with marked pulsation of the abdominal aorta, which is sometimes obvious both to the patient and the doctor. There is in this affection great local discomfort, and even pain, with marked pulsation, obvious to both inspection and palpation. The diagnosis from aneurysm rests partly on the fact that the pulsation is not limited to any part of the aorta, and partly that such rapid and violent action of the heart is not common in aneurysm.

VIII. The HYPOGASTRIC REGION is the situation whence BLADDER, UTERINE, and OVARIAN and TUBAL TUMOURS grow. *Ovarian tumours* (which are nearly always cystic) are usually characterised in the *early stages* by their free mobility, unless they are malignant, and their rapid growth (§ 210). *Tumours of the bladder* are usually rendered sufficiently obvious by changes in the urine. *Tumours of the uterus* are similarly revealed by uterine symptoms, excepting perhaps some subperitoneal fibroids. These may reach a large size without any symptoms at all; their origin and relations are readily detected by bimanual examination. *Pregnancy* causes a symmetrical enlargement, starting from the hypogastric region about the third month of gestation (§ 355). Among the rarer tumours in this region pelvic hydatid and pelvic hæmatocele may be mentioned.

The NATURE, PROGNOSIS, and TREATMENT of these various abdominal tumours are discussed under the organ with which they are connected.

§ 213. **Flattening or Recession of the Abdomen** is not a sign of any great importance. " Ventre plat, enfant il y a," is a French expression signifying that the abdominal wall slightly recedes during the first two or three months of pregnancy. It is met with in abstinence from food, and in wasting disorders, such as cancer and tubercle. It may be present also in intestinal, hepatic, and renal colic, and it may occur as a consequence of excessive purging or vomiting. A hollow or " boat-shaped " abdomen is said to be characteristic of meningitis in infants. It may' also occur when acute general peritonitis is present,-especially in children.

CHAPTER X

THE STOMACH

Two features strike the student in this department of medicine. First, that we are largely dependent upon subjective symptoms in the investigation of disorders of the stomach, a large proportion of the disorders of this organ being functional. Until the use of the "test-meal" (§ 223) and methods of examination by X-rays were adopted, we had to rely almost entirely upon the patient's sensations before and after meals to form a diagnosis. The other feature relates to the important and widespread effects which derangements of the stomach produce in the general economy. The nutrition, of course, fails ; but, apart from this, sufferers from gastric disorders are always liable to mental depression, which may sometimes be extreme. Prostration may occur in all acute abdominal diseases ; but in chronic disorders of the stomach the functions of the nervous system may be so profoundly disturbed by neurasthenic and other symptoms that the physician may overlook the primary cause of the mischief—namely, malassimilation of food.

The digestive system is influenced by two sets of nerves—the sympathetic and vagus ; their relationship and equilibrium may be disturbed by (1) reflex conditions, (2) asthenia of the nervous system, and (3) by emotions. Many symptoms hitherto regarded as functional have been found to be associated with organic disease, and conversely, many hitherto regarded as organic are now known to be frequently of functional origin. Thus it is now realised that dilatation of the stomach is not only of organic origin ; it may come and go with conditions of nerve strain and overwork. Mental and emotional strain can cause profound changes in the secretion of hydrochloric acid in the stomach. On the other hand, the so-called acid dyspepsia, or hyperchlorhydria, is sometimes due not to any organic change in the stomach itself, but to a reflex from a lesion in another part of the abdomen. Gastric symptoms indeed are frequently not of gastric origin, but reflexly associated with diseases of the heart, kidney, gall-bladder, duodenum, appendix, or internal secretory glands.

PART A. SYMPTOMATOLOGY

The symptoms which reveal disorders of the stomach may be local (viz., epigastric pain or discomfort, nausea or vomiting, hæmatemesis,

dryness or bad taste in the mouth, flatulence, heartburn, water-brash, thirst, altered appetite) ; or **general** and **remote** (viz., cardiac symptoms, various nervous derangements, skin symptoms, and emaciation).

Among the **Local Symptoms** of gastric disorder, PAIN or DISCOMFORT AFTER FOOD, and NAUSEA or VOMITING, are perhaps the most constant and important—*i.e.*, the cardinal symptoms. HÆMATEMESIS is less frequent, but more serious. The other local symptoms are also of much value for diagnostic purposes. '

§ **214. Gastric Pain,** or discomfort, in diseases of the stomach, is a most important *local* feature. Although it is not in every case sufficiently constant in its characters to enable us to establish the diagnosis, nevertheless it merits the closest study. In some cases it is altogether absent (even when simple ulcer or malignant disease exists), but when present, the features which should be noted are its *position*, its *character*, its *degree*, its *constancy*, and above all, its *relation to the taking of food*.

Its *Position* is usually over the epigastrium, but pain is very frequently complained of between the shoulders, and very severe pain in the back may also occur. A very localised pain with tenderness is characteristic of ulceration. In its *character* it varies considerably. Sometimes it is like a dull weight or a feeling of distension, such as occurs in atonic dyspepsia and chronic gastritis ; or it may be of a burning character, as in acid dyspepsia ; or it may resemble abdominal cramp, as in spasm of the pylorus, or in some cases of gastralgia. Sharp or lancinating pain of a continuous character usually attends ulcer or cancer of the stomach.

Its *Relation to Food* is by far the most important feature of the pain in gastric diseases : (*a*) *It comes on at once* and lasts a variable time in atonic dyspepsia, in acute gastritis and in ulcer (simple or malignant). In simple ulcer the pain is at once relieved by vomiting—a very characteristic feature ; and solids usually give more pain than liquids. In gastric ulcer the sequence generally is food, ease, pain, ease till food is taken again. (*b*) When pain *comes on an hour or more after food*, it is due to excessive acidity, either from hypersecretion or fermentation (organic acids). In hypersecretion, pain is relieved by taking food and alkalies. Pain coming on late after food is frequent in duodenal ulcer (hunger pain), and the sequence tends to be food, ease, pain lasting until the next meal. A similar pain may be caused by chronic appendicitis or gall-stones. (*c*) Pain *coming on without time relation to food* is characteristic of gastralgia, and is met with in carcinoma of the stomach.' If pressure *over the seat of pain* relieves it, the condition is probably functional, not organic. '

Fallacies.—Pain of the acute type met with in gastralgia may be mistaken for *biliary colic*, but in that condition the pain is greater on the right side, and is sometimes followed by jaundice. In *hepatic* disorders, pain is more often limited to the right hypochondrium. The spine should always be examined for *caries*, especially when stomach pain is complained ' of by children. The pain in such cases is referred to the terminations of

the intercostal nerves. The gastric crises of *tabes dorsalis* may be mistaken for simple gastritis. Pain in the *chest* (§ 25) must not be mistaken for stomach pain. *True angina pectoris* might be mistaken for that type of dyspepsia in which the stomach is distended with gas and hampers the heart's action. Darting or lancinating pain may be due to *growths* involving the nerves near the stomach. In acute *pancreatitis* there is extreme pain of sudden onset in the left hypochondrium, and the case *usually terminates fatally in a few days. Other pancreatic diseases are also attended by pain in the situation of the stomach.

§ 215. **Nausea** or **Vomiting** is, after pain, the most frequent and most definite symptom of stomach disorders, though it arises, also, in many other conditions. Its causes may be grouped under three headings : (*a*) Local Causes ; (*b*) Nervous Causes ; and (*c*) Toxic Causes. Waterbrash (*vide infra*) is sometimes spoken of by the laity as "vomiting," but is, not true vomiting. Regurgitation from a dilated œsophagus is another fallacy.[1] The mechanical discomfort of prolonged coughing may induce vomiting. Phthisical patients may come complaining only of the vomiting, and the physician may be led in consequence to treat the stomach instead of the lungs.

(*a*) LOCAL CAUSES, producing vomiting, include : (1) *Errors of diet*, such as shell-fish, tinned food, excess of alcoholic and other irritating foods. Under these circumstances the vomiting of the peccant material occurs soon after ingestion. (2) *Irritant and corrosive poisons and emetics* also speedily give rise to vomiting. The diagnosis of this cause is aided by (i.) an examination of the vomit, which should *always be preserved ;* it may smell of phosphorus (which is luminous in the dark), or of carbolic, or other acids. (ii.) An examination of the mouth for any corrosive action. (iii.) The occurrence later of the toxic effects peculiar to the several poisons ; and (iv.) a history of poisoning obtained from the patient or his friends. (3) *Fermentation* of the contents of the stomach, such as that met with in dilatation, when the vomiting may occur at very considerable intervals, sometimes of a day or two ; the vomited matter also. is frothy, and contains sarcinæ and yeasts. (4) *Diseases* such as acute gastritis, cancer, and simple ulcer are usually accompanied by vomiting. In chronic gastritis mucus is vomited chiefly in the early morning.

(5) With *dilatation of the duodenum*, vomiting occurs some hours after meals and when lying on the left side. Pain may be absent ; vagotonic symptoms are usually present.

(6) Persistent vomiting and marasmus in young infants are the two chief symptoms of **Congenital Hypertrophic Stenosis**. The symptoms begin about the end of

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[1] How closely regurgitation from the œsophagus, especially when it is dilated, may simulate vomit from the stomach is evidenced by three cases narrated by Dr. J. S. Bristowe (" Clin. Lects. and Essays on Dis. of the Nervous Syst.," pp. 42 *et seq.*). The chief differential features are the ease and promptness with which food is returned from the œsophagus in cases of dilatation and spasm, the *absence of an acid reaction* in the matters so returned, and the absence of signs or symptoms definitely referable to the stomach.

the second week of life—(i.) forcible vomiting, which cannot be stopped ; (ii.) progressive emaciation ; (iii.) constipation ; and later (iv.) visible peristalsis of the stomach. (v.) A small hard nodule (the hypertrophied pylorus) may be made out under the upper part of the right rectus. Careful feeding, lavage, and atropin gradually increased to the toleration limit, sometimes effect a cure. Rammstedt's operation gives good results, but it must not be left as a last resource.

(b) VOMITING OF NERVOUS ORIGIN may be classified under two groups —(a) from cerebro-spinal irritation, and (β) reflexly from visceral irritation.

(a) That due to CEREBRO-SPINAL IRRITATION. 1. In *Hysterical Vomiting* the vomiting may follow any or every kind of food, no matter what its quantity or quality may be ; or perhaps digestible articles like milk will cause vomiting, while indigestible things like cheese may be retained. Sometimes this vomiting resembles a simple regurgitation, as compared with the urgent vomiting of organic disease, the symptoms of which are wanting.

2. In *Migraine* and *Bilious Headache* the patient perhaps awakens with a headache, and vomits only bile (merely an indication that the vomiting is urgent, or that the stomach is empty) ; the headache being relieved by the sickness.

3. Another important cause of vomiting is *Cerebral Disease—e.g.*, tumour, early meningitis, abscess, Ménière's disease. This is recognised by : (i.) The vomiting occurs without relation to food ; (ii.) there is no nausea ; (iii.) the vomiting may be excited by simple change of posture ; (iv.) the presence of other cerebral symptoms, such as vertigo and perhaps optic neuritis (Chapter XIX). Vomiting may also attend the gastric " crises " of *locomotor ataxy*. It occurs at intervals, and is usually severe. It is recognised by the absence of the knee-jerk and the presence of other symptoms of the disease. Vomiting may be associated with *glaucoma*, which is easily overlooked.

(β) Reflex vomiting from VISCERAL IRRITATION may be met with in a great many abdominal disorders, such as peritonitis, pancreatitis, intestinal, biliary, or renal colic ; in all stages of intestinal obstruction, in strangulated hernia, and with intestinal new growths. In the last named the attention of the physician is often drawn from the true source of trouble. It occurs also with pregnancy, uterine and ovarian disorders. If at the end of an operation the surgeon puts in stitches while the patient is coming out of the anæsthetic, vomiting is at once excited every time the needle is put in ; this is especially noticeable with children. Pharyngeal irritation, especially in alcoholics and smokers, leads to prolonged hawking often succeeded by vomiting.

(c) TOXIC CAUSES are uræmia, Bright's disease, and jaundice. Some of the acute specific fevers are accompanied by vomiting, especially at their advent. The vomiting of Addison's disease, hyperthyroidism, and pernicious anæmia comes under this heading. Certain cases of vomiting in pregnancy are due to toxæmia ; the urine shows a higher ammonia coefficient than with reflex vomiting. After anæsthetics vomiting may

be urgent; sometimes this is due to blood in the stomach, and will cease when it is expelled.

The *Treatment of vomiting must be directed to its cause*, but there are certain measures which can be applied to relieve the symptom. The patient should be kept absolutely at rest in the horizontal position, and without food, or only given milk in small quantities at a time, and iced water. Milk diluted with barley-water, whey, or peptonised milk are given where ordinary milk is not retained. Among the remedies which may be employed are effervescing mixtures, alkalies, hydrocyanic acid, bismuth, drop doses of vinum ipecacuanha or tr. iodine (one drop in a teaspoonful of water hourly), opium, and acetanilid (especially in the vomiting after anæsthetics), sod. bicarb. Зi (4) to Oi (500) water, seidlitz powder (if the vomiting be due to constipation) or calomel. Bromides and hydrocyanic acid are useful for nervous vomiting; a mustard-leaf applied to the epigastrium may also be useful. Washing out the stomach with warm water or normal salt solution often gives relief. For *Sea-sickness*, chlorobrom, bromides, morphia, and recently chloretone and validol are recommended very highly.

Cyclical or Recurrent Vomiting is an occasional condition occurring in children. Usually two or three attacks occur in the course of a year; they last a few days to two weeks, and come on without assignable cause, even with the most careful dieting. An attack comes on suddenly with drowsiness, constipation, and perhaps pyrexia; all food is vomited; the breath smells sweet from the presence of acetone, and acetone and diacetic acid are found in the urine (*vide* § 313). Drowsiness and restlessness increase, and cases have been mistaken for meningitis and intestinal obstruction. The condition is that of Acidosis, probably due to defective function of the liver (§ 323). It is important to open the bowels freely. Copious enemata with normal saline, and by the mouth frequent small doses of alkaline carbonates and citrates may be tried.

§ 216. Hæmatemesis (Vomiting of Blood).—Bleeding from the stomach, unless in slight quantity, is usually accompanied by nausea and vomiting. In the first place, it is important to decide whether the blood really comes from the stomach and œsophagus.

Sources of Fallacy.—(1) Blood from the lungs may be mistaken for blood from the stomach (see Hæmoptysis, § 84). (2) *Epistaxis*, the blood running down the gullet and being vomited, is a common fallacy in children, in whom the blood is apt to be swallowed. The same may follow operations on the tonsils or teeth. Epistaxis is recognised by making the patient blow his nose. In epistaxis there are no abdominal symptoms. (3) Blood from the *fauces* or *gums*, especially when the gums are spongy, or when pyorrhœa alveolaris exists, may give rise to a sanguineous vomiting or expectoration, the cause of which is very apt to be overlooked, if unsuspected, even by competent observers (§ 169); but the blood is mixed with saliva, and is rarely very large in amount. (4) Blood from a fracture of the base of the skull and from œsophageal disease

may also be swallowed and vomited. On the other hand, *hæmorrhage from the stomach* is (i) preceded by a feeling of faintness and nausea, and (ii) followed by melæna (tarry stools). (iii) Blood from the stomach is mixed with food, and mostly brown (" coffee-grounds "), though it may be red if the quantity is large (*e g.*, in ulcer) or if food has been brought up before the blood. (iv) There is an absence of previous history or local signs of pulmonary disease, and there may be a previous history of disease or derangement of the stomach or liver.   •

The *Causes of Hæmatemesis* may be roughly divided into (*a*) those which produce a slight or protracted hæmorrhage, and (*b*) those which give rise to a large quantity at one time.

' (*a*) **Slight or Protracted Hæmorrhages** occur chiefly in Chronic Gastritis and Cancer. A temporary irritation or congestion of the stomach produced by irritating articles in the food or by urgent vomiting, may be attended by streaks of blood in the vomit.

I. CHRONIC GASTRITIS, or gastric catarrh, is known by (i.) vomiting in the morning—often viscid mucus streaked with blood—or at other times. (ii) It may be accompanied by, and due to, disease of the liver (cirrhosis), or advanced cardiac disease, and is found especially in alcoholic subjects (see § 233).

II. CANCER OF THE STOMACH OR ŒSOPHAGUS is recognised by : (i.) The patient is usually beyond middle age ; (ii) pain is complained of—severe, constant, and generally worse after food ; (iii.) the blood vomited is rarely copious, but typically " *coffee-ground* " in character, and may continue for weeks ; (iv.) the hæmatemesis is followed by melæna unless the blood is scanty, and occult blood is usually present in the fæces ; (v.) progressive cachexia is marked, and an abdominal tumour, or evidence of cancer elsewhere, *may* be found (see also § 232).

(*b*) A **Large Hæmorrhage** at one time may occur in Simple Ulcer of the Stomach or Duodenum, Liver Diseases, other diseases giving rise to Portal Obstruction, Aneurysm of the Aorta, Vicarious Menstruation, Gastrostaxis, Morbid States of the Blood, or after taking Chemical Irritants.

III. SIMPLE ULCER [1] OF THE STOMACH.—This is known by : (i.) The hæmatemesis is copious ; therefore the blood is bright red, after first being a little black, and melæna usually follows ; (ii.) characteristic pain occurs directly after food, and is relieved by vomiting ; (iii.) it is found chiefly in young women, (iv.) who are the subjects of anæmia, but not often great emaciation ; (v.) a history of previous attacks of bleeding is often present (§ 231).   •

III*a*. ULCER OF THE DUODENUM is often difficult to distinguish from gastric ulcer. It occurs mostly in men (§ 231). Gall-stones ulcerating into the duodenum may occasionally cause hæmatemesis.   •

IV. LIVER DISEASE (by causing portal obstruction), especially CIRRHOSIS

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[1] Cases have been recorded of profuse hæmatemesis resembling that of simple ulcer, occurring in older patients, which disappeared under antisyphilitic treatment. The condition was apparently a syphilitic ulcer of the stomach.

(§ 275). The hæmatemesis may be slight, but it is more often very copious—the most copious met with.

V. OTHER CAUSES OF PORTAL OBSTRUCTION (see § 209)—*e.g.*, tumour pressing on the portal vein. This, as with cirrhosis, is known by the other symptoms of such disease—*e.g.*, the accompanying and rapidly increasing ascites, splenic enlargement, jaundice, and diarrhœa. Thrombosis of the portal vein is rare and hard to diagnose. It gives rise to sudden onset of signs of portal obstruction.

VI. ANEURYSM OF THE AORTA, or of one of its branches, leaking into the bowel, or œsophagus. This is known by (i.) possibly a previous history of aneurysmal symptoms (§ 65); (ii.) the blood is copious; (iii.) sudden death is the usual result. This is the usual course, but in certain other cases there is a small recurrent leakage from the aneurysm for a few days or weeks preceding death.

VIII. VICARIOUS MENSTRUATION.—It is impossible to be certain in the diagnosis of this condition. It occurs periodically, and in women with amenorrhœa.

VIII. GASTROSTAXIS.—Under this title are included cases of severe hæmatemesis, occurring usually in young anæmic women, due to capillary oozing. Such cases were formerly thought to be due to gastric ulceration, but more frequent operations and post-mortem examinations have shown that no ulcer is present.

IX. MORBID CONDITIONS OF THE BLOOD, such as yellow fever, malignant forms of the specific fevers, purpura, leukæmia, and hæmophilia.

X. CHEMICAL IRRITANTS (*e.g.*, arsenic, strong alkalies, and mineral acids), or mechanical injuries from articles which have been swallowed.

XI. SPLENIC ENLARGEMENT, apart from liver disease or leukæmia, may cause hæmatemesis.

XII. Occasionally, hæmatemesis occurs with CHRONIC NEPHRITIS.

In the *Differentiation* of the causes of hæmatemesis (1) examine the stomach; (2) examine the liver, especially for CIRRHOSIS, which is perhaps the commonest cause of hæmatemesis, simple or malignant ulcer being the next; (3) examine the chest for aneurysm or other mediastinal growth which may have perforated the œsophagus; (4) ascertain the approximate quantity of vomited blood, and then review the case.

Prognosis.—Hæmatemesis is usually a serious symptom, but its gravity depends upon the cause. In portal congestion, hæmatemesis not infrequently serves as a safety-valve to relieve the abdominal congestion, and in a sense is beneficial. As regards the lesion, aneurysm is the most grave of the causal conditions; then, in order, cancer, morbid blood states, cirrhosis, and simple ulcer. The amount of hæmorrhage is a less valuable guide to prognosis, although where the amount is copious the patient will remain debilitated for a considerable time.

The *Treatment* of hæmatemesis must also have reference to the cause. (a) When small in quantity, it calls for but little immediate treatment. (b) When in larger amount, the patient should not be moved from the

place where the bleeding occurred, but must be kept absolutely at rest in the horizontal position. An ice-bag should be placed over the epigastrium. The distressing thirst may be relieved by allowing the patient to suck a linen rag moistened with water. Nothing should be given by the mouth except iced water for some time (see Gastric Ulcer, § 231). Morphia hypodermically is the best hæmostatic, and also relieves anxiety and pain. If bleeding continues, give astringents by the mouth, such as alum, gr. v. (0·3), and dilute sulphuric acid, ℥xx. (1·2), or ergot. Adrenalin (1 in 1,000), ½-drachm (2) doses every two or three hours, is a valuable remedy. Calcium chloride gr. 1 (0·65) in 100 minims of sterile water may be injected. Normal serum is also very useful. Coagulose is of use when the blood coagulation time is delayed. In profuse hæmorrhages subcutaneous infusion of saline may be necessary. Gum açacia 6 per cent. in 0·9 per cent. salt solution may be given intravenously, or blood transfused from a suitable donor.

§ 217. The **other Local Symptoms** of gastric disorder are of considerable diagnostic value.

1. BAD TASTE IN THE MOUTH is very often complained of in gastric disorders, and is always most noticeable in the morning. DRYNESS OF THE LIPS is another very constant manifestation, and will often give an acute observer the first clue to the existence of gastric disorder.

2. THIRST is often associated with dyspepsia ; it is specially apt to occur with dilatation of the stomach, inflammatory stomach lesions, and in all cases of persistent vomiting.

3. FLATULENCE is a distension of the stomach or intestines by gas, which may be brought up by the mouth or passed by rectum. This gas may be due to repeated swallowing of saliva and air, or to decomposition of food. Among its causes are excessive ingestion of vegetables, sugars, and starches, chronic dyspepsia, chronic gastritis, and all conditions attended with dilatation of the stomach. Semi-voluntary swallowing or gulping down of air (aerophagy) is met with in lunatics, and in some hysterical or neurotic individuals without gastric derangement. It is diagnosed from dyspepsia by the absence of all other symptoms of that condition.

4. " HEARTBURN " and ACID ERUCTATIONS are usually met with together. Heartburn is a burning sensation passing up from the epigastrium to the pharynx, and sometimes mouthfuls of acid fluid are brought up at the same time. It is due to hyperacidity and partial regurgitation of the gastric contents into the lower end of the œsophagus. The treatment of 3 and 4 is discussed in § 228.

Causes.—Hyperacidity, or " acid risings," may be of two kinds. (a) *Organic acids* are met with in diseases where there is *deficient* gastric secretion—some forms of atonic dyspepsia, chronic gastritis, cancer, and dilatation of the stomach. HCl is a germicide, and when from any cause it is absent, bacteria flourish ; fermentation ensues within a *few hours* after food, and is accompanied by pain in the epigastrium. The three

principal types of acid fermentation are : butyric, lactic, and acetic.

(b) Hyperchlorhydria, or excessive secretion of HCl, is met with in one form of acute dyspepsia, and is usually present with gastric and duodenal ulcer. The name is often misapplied to the acidity due to organic acids arising from fermentation. Here, the pain or " gnawing " occurs before meals, and is temporarily relieved by food (see also § 229).

5. HICCOUGH.—Normally the opening of the glottis synchronises with the contraction of the diaphragm, and consequently there is no hindrance to the free entry of air. Hiccough is caused by a spasm of the diaphragm which occurs at irregular intervals and sometimes at the moment of closure of the glottic aperture. The characteristic cough is then heard. The important causes of persistent hiccough are : (1) Reflex stimulation of the phrenic nerves by gastric flatulent distension or irritation after hot, peppery foods and with hepatic disease. (2) Irritation of the peritoneum, as in peritonitis, general or local, near an inflamed abdominal organ, or in typhoid fever. (3) Disease of the thoracic viscera, especially diaphragmatic pleurisy. (4) Toxic blood conditions, notably uræmia. (5) Neurosis. To this cause are assigned certain cases for which no more adequate reason is apparent. Hiccough may also occur as a symptom of hysteria, of cerebral tumour, meningitis and encephalitis lethargica.

Prognosis.—Hiccough is not as a rule a symptom of any great significance. In abdominal disease its occurrence is of grave import. Occasionally it may be persistent and resist all treatment ; it exhausts the patient, and may be the immediate cause of death.

Treatment.—The simplest forms of treatment are those directed to producing definite physiological contractions of the diaphragm. These are such well-known methods as sipping water and holding the breath. Anything which gives rise to a feeling of suffocation may cause a forcible contraction of the diaphragm, and so stop the spasm ; for this reason tickling the nares and taking snuff have been tried, often with success. Dyspepsia is the most common cause in operation, and the hiccough is readily cured with bicarbonate of soda and peppermint. If these measures do not suffice, or if the hiccough recurs frequently, a thorough investigation of the patient is called for. When no causal condition can be found and the hiccough continues to be severe, one may give sedative drugs by the mouth, or, if necessary, by the rectum ; the bromides and tinct. opii or $\frac{1}{10}$ gr. (·006) apomorphine subcutaneously are successful. Peripheral stimuli, such as blisters to the epigastrium, pinching the lobe of the ear, forcible pulling forward of the tongue, and digital pressure on the vagus in the neck, may be tried ; and the abdomen may be bound tightly with a bandage or with plaster. Chloroform may have to be administered. Of drugs, the opiates, those of the acetanilide group and the nitrites have most often been of use.

6. " WATER-BRASH " (Pyrosis) is the name given to a clear alkaline fluid expelled from the mouth in gushes, most often in the morning. Sometimes it is expelled without any kind of straining, but more often

it is attended by retching. It is probably a reflex hypersecretion of saliva due to irritation in the stomach, swallowed during the night. It is met with in many dyspeptic conditions, and is a fairly constant symptom in chronic gastritis.

7. ANOREXIA (Loss of Appetite) is not always an indication of stomach disease, as it is present in many general constitutional disturbances. Its chief clinical importance lies in its presence in the earliest stage of gastric cancer. In atonic dyspepsia there is sometimes no appetite before a meal ; but the first few mouthfuls of food induce secretion of gastric juice, and so excite appetite. HYSTERICAL ANOREXIA (Anorexia Nervosa) is known by : (i) The appetite is perverted ; for instance, the patient will only eat some unreasonable article—e.g , a bun bought at a particular shop. Such patients may push matters to extremes, almost to the point of death. The condition is really a form of hysterical insanity. (ii.) It is only met with in the female sex, and (iii.) careful investigation excludes any organic condition.

INCREASED APPETITE is far more often met with, as Shakespeare pointed out, in gastric disorders. It is found in some cases of chronic dyspepsia, chronic gastritis, and dilated stomach, in pregnancy, and during con-valescence. A FALSE APPETITE which is satisfied with the first few mouth-fuls of food is sometimes met with in subacute and chronic gastritis, owing to the irritated condition of the mucous membrane and in hyptonic dyspepsia. BULIMIA or ravenous appetite is seen in diabetes, in neuroses of the stomach, after acute gastritis, in wasting disorders such as mesen-teric gland disease, in phthisis, intestinal worms, and Graves' disease. PERVERTED APPETITE, excessive fondness for acids and sweets, or desire to eat objects such as chalk, pencils, or hair, may occur in hysteria, chlorosis, and pregnancy. A FOUL BREATH is present in some forms of gastritis. It has been proved to be due in some cases to an infection of the stomach wall by streptococci and B. coli. It is more often due to want of cleanliness of mouth and teeth.

§ 218. General or Remote Symptoms are very constant accompaniments of all gastric diseases.

1. GENERAL MALAISE and a sense of ill-health and incapacity for work are among the earliest and most constant accompaniments of all derange-ments of the digestion, whether functional or organic. The dark rim beneath the eyes, and the sallow, " earthy " complexion, so frequently associated with town-dwellers, are quite as often due to dyspepsia, just as this latter is often due to defective teeth or to the insufficient use of them. EMACIATION is not so frequently associated with gastric disorder as might be supposed, though in very chronic cases there is sure to be some loss of flesh. Early and marked emaciation is, however, one of the surest indications of cancer of stomach.

2. The CARDIAC SYMPTOMS met with in dyspepsia are palpitation, pain in the region of the heart (pseudo or reflex angina) ; dyspnœa, syncope, and vertigo ; intermission of the cardiac rhythm ; and cough, due to

pharyngeal catarrh or reflex irritation. Collectively, these symptoms may, as previously mentioned, give rise to the impression that the case is one of cardiac valvular disease, although the heart may be structurally healthy.

3. FUNCTIONAL DISTURBANCE OF NERVOUS SYSTEM.—*Headache and depression of spirits* are invariably met with in all forms of dyspepsia. A sense of general ill-health and irritability of temper out of all proportion to the local mischief attend most gastric disorders, and, where stomach symptoms are not prominent, may lead the physician away from the true cause. Any or all of the symptoms of *neurasthenia* (Chapter XIX) may undoubtedly result from gastric disorder, and this constitutes one variety of what the author has described as Toxic Neurasthenia.[1]

4. DIARRHŒA may accompany stomach disease when the gastric contents are of an irritating nature ; CONSTIPATION is usually found with simple ulcer, cancer, and chronic gastritis. But a more usual condition is an IRREGULARITY of the bowels, accompanied by borborygmi (rumbling in the bowels).

5. The URINE invariably exhibits signs which reveal the disturbances in the metabolism of the body. The commonest of these, perhaps, is an excess of URATES, as shown by the pinkish sediment when the urine cools. In other cases PHOSPHATES form the deposit ; and in certain cases OXALATES are found (compare § 340). In these circumstances dyspepsia must be regarded as a predisposing cause of renal and vesical calculus.

6. SKIN SYMPTOMS.—General *pruritus* may accompany many forms of gastric derangement. *Flushing* of the face after meals is met with in many gastric disorders, especially when they occur in the female sex. Acne Rosacea is a common skin disease due to dyspepsia. The face may be swollen so that the case appears like one of acute Bright's disease ; but the sudden onset, without much constitutional disturbance, and early disappearance on curing the indigestion, distinguish it from that disease. The occurrence of general *urticaria* in certain individuals after eating indigestible articles is very common. It 'may also attend the different forms of gastric disorder.

PART B. PHYSICAL EXAMINATION

Disorders of the stomach are investigated by Inspection, Palpation, Percussion, Auscultation, X-ray examination after an opaque meal, and by Examination of matters vomited, or withdrawn from the stomach by a tube.

§ 219. Inspection.—(1) The *Teeth* in all cases must be closely examined. Common causes of indigestion are defective teeth, oral sepsis and bolting the food. Disorders of the teeth are referred to in § 166 and § 169.

(2) The *Tongue* and its diseases have already been described, and

[1] " Clinical Lectures on Neurasthenia," Glaisher, London.

O.M.

§ 171 should be specially consulted. At one time the tongue was thought to indicate the state of the stomach, but this is by no means always the case, and it is a far more certain indication of the patient's general condition. But even in this, allowance has to be made for certain variations —namely : (i.) Individual variations, since a coated tongue is normal to some, even in health, and a clean tongue in others may be associated with disease ; (ii.) certain diets—*e.g.*, milk—produce a coated tongue ; and (iii) certain habits—*e.g.*, smoking and "tippling"—also coat the tongue. The mouth may show signs of poisoning by corrosive acids.

(3) Inspection of the epigastric region may reveal a tumour, or the peristaltic movements of a dilated stomach. Aortic pulsation may be transmitted by a pyloric tumour, although no bulging is visible.

(4) In skilled hands the œsophago-gastroscope may be employed to examine the interior surface of the stomach.

§ 220. **Palpation.**—To palpate the stomach successfully requires considerable experience. The patient's shoulders should be supported and his arms relaxed by the sides, and he should be instructed to open his mouth, to draw up his knees, and to " let his breath go." [1] Talking to him is useful to distract his attention. The hand should always be warmed, and it should then be laid quite *flat* upon the abdominal wall. Then only can be detected the presence of a tumour, tenderness, or other abnormality.

Gastric Succussion or *Splashing* is made out by placing one hand on each side of the stomach, and suddenly pressing inwards the finger-tips of each hand alternately. Listening over the stomach with a binaural stethoscope during this procedure materially aids in discovering this sign Splashing can be *normally* elicited during the process of digestion—*i e* , during the first hour or two after a meal, especially if much fluid has been taken. But if succussion can be elicited after that time, it suggests that there is atony of the stomach, either with or without dilatation.

§ 221. **Percussion** of the stomach is not very satisfactory or precise. **Surface Anatomy of the Stomach.** (Figs. 59 and 71.) The cardiac orifice lies behind the seventh left costal cartilage 2¼ inches from the midline. The fundus occupies the left dome of the diaphragm and lies behind the apex of the heart. As this part of the stomach always contains gas, it is resonant (Traube's space). The body of the stomach is vertical, and turns sharply into the pyloric antrum. The pylorus lies opposite the first lumbar vertebra in the transpyloric plane just to the right of the midline. The lesser curvature is extremely variable and depends on the state of filling ; it may reach below the umbilicus in normal conditions. The rough outline of the stomach resonance may be defined after giving successively the two portions of a seidlitz powder dissolved

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[1] Some say it is better to have the legs extended loosely, and some advise examination in a hot bath to relax the muscles. Finally, anæsthesia with chloroform or ether may be necessary in very obscure cases.

separately. In this way dilatation (§ 234) may be distinguished from gastroptosis (§ 235).

§ 222. The Motor Functions of the Stomach and Intestinal Tract are most accurately investigated by X-ray examination after an opaque meal.

The presence of food from a previous meal in the syphonage from a test-meal may indicate delay in the stomach. After a normal meal consisting of 50 grammes of bread, 200 grammes of beefsteak and a glass of water, no solid portions should be found in the stomach in six or seven hours. There is, however, considerable individual variation. Delay in the stomach may be tested by giving a teaspoonful of charcoal the night before a test breakfast. Charcoal so given should appear in the faeces also in thirty-six to forty-eight hours. If it does not appear on the second morning, an enema should be given. The presence of charcoal in the returned enema shows delay in the lower colon ; if it is not present, the delay is higher up. This test is not very accurate.

X-ray Examination is carried out with the fluorescent screen after giving 3 ounces of barium sulphate suspended in gruel, porridge or buttermilk. Radiograms taken can be studied afterwards. The meal is seen passing down the oesophagus and any obstruction or diverticulum is noted. The outline, position, tone and the rate and character of the peristaltic movements of the stomach are observed, the time at which it is empty, and the passage through the pylorus and duodenum. Irregularity of outline may be seen if there is a growth of the stomach, and if the barium lodges in the crater of an ulcer. The normal shape of the duodenal cap is characteristic, and is altered by ulcer, adhesions or pressure from without, as by a distended gall bladder. The position and mobility of the lower ileum and caecum are observed, and the appendix may be seen filled. The passage of the material through the colon is watched at intervals. Normally the stomach empties in three-and-a-half to five hours ; the terminal ileum and caecum commence to fill about the same time. The terminal ileum should be clear of material four hours after the stomach is empty, and the colon should be clear in seventy-two hours.

§ 223. Examination of Stomach Contents.[1]—First, as to the CHEMISTRY OF DIGESTION, from a clinical standpoint, and the practical information to be derived from clinical examination of the stomach contents. *Four processes* normally take place in the stomach : (1) The conversion of starch into sugar, begun in the mouth, is carried on a stage farther ; (2) proteins are changed into peptones ; (3) fat globules are set free from their envelopes ; (4) milk is curdled. Delay in digestion may be caused by (1) deficient peristalsis of the stomach walls, (2) deficient quality or quantity of the gastric juice,) the consumption of indigestible articles, or (4) the dilution of the gastric juice by drinking too much fluid at meal-time.

The gastric juice contains HCl, water, pepsin, rennin, mineral salts, and a *little* mucus. Pepsin and rennin exist in the secretory cells only as zymogens, which, in the presence of the HCl, become active ferments or enzymes. In the healthy state, as the result of digestion, about 30 c.c. of fluid should be obtained from the stomach one hour or so after a test-meal (*vide infra*), straw-coloured, without much odour, without organic acid, and with about 0·2 per cent. of free HCl.

As regards *hydrochloric acid*, much depends on the time of examination. *Hyperchlorhydria* is merely a convenient term for excessive secretion of gastric juice. It has come to be somewhat loosely used for " excessive acidity," and thus to be confused with the acidity of fermentation (due to organic acids). On the other hand, after a meal, a negative result on testing for HCl would indicate the absence of peptic activity, as an acid is required to convert the inactive proenzyme or pepsinogen into pepsin. An excess of HCl is distinctive of gastric and duodenal ulcer, as compared with gastralgia ; for in the latter the HCl is normal or diminished. HCl is also diminished in all catarrhal conditions of the mucous membrane, in great anaemia, and in states

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[1] It is not possible here to give more than a brief outline of this important subject.

of nerve exhaustion. When there is a difficulty in diagnosing malignant disease, the absence of free HCl is a point in favour of cancer.

Lactic acid is not normally present in the gastric juice after digestion has proceeded for one hour, but traces may be found, due to the ingestion of lactic acid in certain foods, or to fermentation in the mouth. A decided reaction with Ueffelmann's test (infra) is found with cancer of the stomach, but a negative reaction does not have equal value in proving the absence of the disease.

Butyric and acetic acids prove the presence of fermentation, and are found where HCl is deficient, or the food is delayed in the stomach, as in dilatation of the stomach, or a narrowing of the pylorus.

The secretion of pepsin is not interfered with, unless there be destruction of the glandular elements of the stomach. The pepsin varies with the hydrochloric acid. An acid secretion without peptic activity does not, I believe, occur.

Renninogen and Rennin are diminished or absent in the later stages of gastritis and cancer. The amount of rennin present appears to be directly proportional to the quantity of pepsin.

Examination of Gastric Contents after a Test-meal is a useful method of investigating the secretion and motility of the stomach. It consists of three steps : (a) The administration of the test-meal ; (b) the withdrawing of the gastric contents by means of a stomach-tube ; and (c) the microscopical and chemical examination of the material withdrawn.

(a) The test-meals suggested are of many kinds. The usual test breakfast is 2 ounces of toast and 10 ounces of tea with milk. This is given in the morning before any food is taken, and drawn off one to one and a half hours later. A test lunch containing 3 ounces of minced beef, 5 ounces of potato and 10 ounces of water should be withdrawn two and a half to three hours after being taken. The following method gives a great deal of information from one examination and is recommended. The patient takes boiled egg, toast and tea at eight o'clock and no more food till the test meal at one o'clock, when he has 3 ounces of bread without crust and a pint of water. This is withdrawn an hour later. Portions of egg and toast are easily recognised and if present indicate delay in the stomach.

(b) Method of Passing the Tube.—Use a rubber tube nearly 2 yards long, with large "eyes." It should be thoroughly cleaned, and moistened with warm water. The patient should sit with the head erect, the chin being projected forward, and the mouth open. The patient is told to draw long breaths in and out; in this way retching is inhibited. The tube is passed into the pharynx, while the patient is instructed to swallow, and the tube is pushed down into the stomach. Then bring the end down to a lower level than the stomach, and the contents should siphon out into a receptacle. If the gastric contents do not flow at once, the patient should strain, as if trying to vomit ; or draw out the tube a little, lest the end becomes folded upon itself, or the "eye" stopped by a fold of mucous membrane. It may be necessary to start the flow by suction with a syringe. The tube should be marked at a position 16 inches from its tip. By this means we know the amount of tube which has been passed into the œsophagus, because when the mark is opposite the incisors, the tip ought to have reached the stomach. A flexible tube curling up in the œsophagus is a contingency which may thus be obviated. Its use is contra-indicated in angina, heart failure, fever and other acute diseases, recent hæmorrhages, great arteriosclerosis, aneurysms, and a high degree of emphysema and bronchitis.

The fractional test-meal yields more accurate information as to the condition of the gastric secretion, the emptying of the stomach and the neutralisation of excessive acid by the reflux of alkaline duodenal contents. A soft rubber tube, of the size of a 6 or 8 catheter, and with an oval perforated bulb at the end, is swallowed by the patient, any resting contents are withdrawn with a small glass record syringe fixed to the end of the tube, and then a pint of test gruel is drunk with the tube in position. The gruel is made with two tablespoonfuls of breakfast oatmeal mixed with one quart of water, and boiled down to one pint and strained. The tube is kept in position

whilst the patient continues his ordinary work. During the next two to three hours, at intervals of a quarter of an hour, about 10 c.c. are drawn up and placed in a numbered test tube. The contents of the test tubes are separately examined. By this method more complete knowledge is obtained as to the rate of digestion and the emptying of the stomach, and of the condition of the secretion of acid, pepsin, mucus, and the regurgitation of duodenal contents as indicated by the presence of bile, than could be gained by a single sample aspirated an hour after the meal. "Anxiety or worry causes delay, and by means of the fractional test meal it has been discovered that with strong emotional states digestion may stand still for the first hour or more, and if the cause be suddenly removed, digestion proceeds rapidly from that moment."

(c) Examination of Stomach Contents after the test-meal.—GENERAL EXAMINATION —Appearance, acidity to litmus paper, smell, consistency, and presence of slimy mucus, bile, or blood, should first be observed.

MICROSCOPICALLY we can detect fat globules, starch cells, vegetable and muscle fibres, and sometimes fatty crystals, leucin and tyrosin, cells of the mucous membrane, torulæ cerevisiæ, or sarcinæ, and pus cells. Epithelial cells may be in excess in carcinoma. The Oppler-Boas bacillus may sometimes be seen on examination under the high power lens.

The stomach contents should be filtered, and the filtrate used subsequently.

CHEMICALLY we have to answer six questions :

(a) Are the contents acid ? This can be roughly detected by the use of litmus paper.

(b) Method of estimating total acidity—i.e., acidity due to hydrochloric acid, organic acids, carbon dioxide, and such acids as react acid to phenolphthalein. Titrate 10 c.c. filtered gastric contents with decinormal solution of caustic soda (prepared free from carbon dioxide), using 1 per cent. solution of phenolphthalein as indicator. Add a drop or two of the indicator to the gastric contents, dilute with two or three times its volume of distilled water, and then run in the decinormal caustic soda solution from a burette till a uniform faint purple-red tint is produced. If 5·5 c.c. of decinormal caustic soda are required for 10 c.c. of gastric contents, the acidity may be calculated in terms of HCl by multiplying the 5·5 c.c. of the soda solution used by 0·00365 × 10. The percentage of HCl actually present is therefore 10 × 5·5 × 0·00365 = 0·2 per cent.

(c) Is free hydrochloric acid present ? Tests : (i.) A 1 per cent. solution of di-methylamido-azo-benzene in alcohol is used. A drop of gastric contents is placed on a white tile, and a drop of the reagent run alongside. A deep pink-red colour is given by free HCl. (ii.) Gunzberg's test : Two or three grains of phloroglucin are mixed in a small evaporating dish with 1 or 2 grains of vanillin, about 1 c.c. of alcohol is added, and then about 1 c.c. of gastric contents. Evaporate on a water-bath, when a bright cherry-red colour indicates free HCl. If much organic acid is present a slight reaction may occur in test (i.), which, though more convenient, is therefore not quite so reliable as test (ii.), which is never given by organic acids. Free hydro-chloric acid is normally present. It is always present in gastric ulcer and hyperchlor-hydria, hardly ever in carcinoma.

(d) What is the amount of active hydrochloric acid ? The " active " hydrochloric acid includes (i.) free HCl ; (ii.) the HCl which is combined with protein and nitro-genous organic bases. It does not, of course, include the inorganic chlorides like NaCl. Estimation.—Two equal volumes of the filtered gastric contents (10 c.c.) are taken. (i.) One portion is made alkaline with sodium carbonate solution and eva-porated to dryness on a water-bath in an evaporating dish. The residue is ignited over a small Bunsen flame until it is well charred and all the organic matter is decom-posed. (ii.) The other portion of the gastric contents is placed in a porcelain eva-porating dish (4½ inches in diameter), and evaporated to dryness on the water-bath, as in (i.). In each case the dish is cooled, and about 60 c.c. of water, 6 c.c. of pure nitric acid, 3 c.c. of 10 per cent. iron alum solution, and 10 c.c. of decinormal silver nitrate solution, added. Decinormal ammonium sulphocyanide solution is run in until a permanent brownish-red tint appears. The active HCl in 10 c.c. of gastric

contents is equal to the difference of the amounts of ammonium sulphocyanide solution used in (i.) and (ii.). *Example.*—If in (i.) 3 c.c. of $\frac{N}{10}$ sulphocyanide solution are used,

and in (ii.) 8 c.c. of $\frac{N}{10}$ sulphocyanide solution are used, then the active HCl in 10 c.c.

of gastric contents is 5 c.c. of $\frac{N}{10}$ HCl. Therefore the percentage is 10 × 5 × 0·00365 = 0·18 per cent. This is the most important estimation, and the amount normally is about 0·15 per cent. *In carcinoma it is usually much below 0·1 per cent. ; in simple ulcer it is usually considerably above 0·15 per cent.* It is the most important estimation in the analyses of gastric contents.

(e) *Is lactic acid present ?* A qualitative test should be made, a weak solution of Ueffelmann's reagent (made by mixing a little 5 per cent. solution of carbolic acid with a few drops of liquor ferri perchloridi) being added to the filtered gastric contents. The development of a distinct yellow colour indicates lactic acid.

(f) *What is the amount of organic acids ?* This is given with sufficient accuracy by the difference between the total acidity (expressed as HCl), and the active HCl as estimated by the method described above. They are in excess in conditions of much fermentation and gastric carcinoma.

PART C. DISEASES OF THE STOMACH, THEIR DIFFERENTIATION, PROGNOSIS AND TREATMENT

§ 224. The **Routine Investigation** of the disorders of the stomach consists of three steps.

FIRST : We must identify the patient's LEADING SYMPTOMS as being referable to gastric disorder (see Part A.).

SECONDLY : Inquire as to the HISTORY, and especially whether the illness came on *acutely* and recently, or whether, as is more usual, it came on insidiously, and has run a *chronic* course. Much depends on the skill and method with which the history is elicited. Inquire particularly as to pain or discomfort and its relation to meals, and as to the other symptoms mentioned in Part A.

THIRDLY : Proceed to the PHYSICAL EXAMINATION, and ascertain whether there be any localised tenderness and pain, and whether any tumour or other abnormality be present.

Classification of disorders of the stomach.

A. Acute Diseases of the Stomach.
　　I. Acute dyspepsia (bilious attack) : *without tenderness* .. § 225
　　II. Acute gastritis : *with tenderness* .. .. § 226
B. Chronic Diseases of the Stomach.
　　(a) WITHOUT TENDERNESS ON PRESSURE : pain less marked.
　　　　I. Chronic atonic dyspepsia .. .. .. • .. .. § 228
　　　　II. Chronic acid or irritable dyspepsia .. .. .. § 229
　　　　III. Gastralgia (gastric neuralgia) .. .. § 230 •
　　(b) WITH TENDERNESS ON PRESSURE : pain a marked feature.
　　　　IV. Simple ulcer .. ..: § 231
　　　　V. Cancer of the stomach .. .. § 232
　　　　VI. Chronic gastritis .. .. § 233
　　(c) DILATATION OF THE STOMACH .. .. , ... § 234

This classification, based on the presence or absence of tenderness, is not very

satisfactory, and each group will be found to contain many exceptions. It is, however, the least unsatisfactory of those clinical classifications possible at the present time. The division of Chronic Dyspepsia into Atonic and Acid is also very unsatisfactory (see footnote, p. 312).

If the patient's symptoms have come on gradually, and lasted a considerable time, turn to **Chronic Disorders** of the Stomach (§ 227).

If, on the other hand, his symptoms have commenced somewhat suddenly and recently, the case is probably one of the two **Acute Disorders** of the Stomach : I. ACUTE DYSPEPSIA ; or, II. ACUTE GASTRITIS.

I. *The patient—whose temperature is normal—complains of* NAUSEA, GASTRIC DISCOMFORT, *headache, and depression, which have come on suddenly ; and there is no marked epigastric tenderness.* The disease is probably ACUTE DYSPEPSIA.

§ **225. Acute Dyspepsia** ("Bilious Attack") consists of a sudden disturbance of the digestion in a previously healthy person, such as occurs in association with surfeit, high living or other errors in diet.

The *Symptoms*, which come on suddenly, are : (1) Pain, or a feeling of oppression or distension in the epigastrium, occasionally accompanied by some tenderness on pressure, though the tenderness is never very marked. (2) Nausea and vomiting very generally ensue (but not always). (3) Headache, depression, anorexia, coated tongue, constipation, scanty urine loaded with urates. (4) The illness is sometimes preceded and accompanied by drowsiness, and not infrequently there is a history of previous similar attacks.

The *Diagnosis* is not difficult, the only condition resembling it being acute gastritis, in which the constitutional symptoms are more apparent, the duration of the illness considerably longer, and the *tenderness much more marked*. Irritant poisoning comes on much more suddenly with very urgent vomiting (§ 215).

Etiology.—(1) Too large a meal, especially after previous fatigue. (2) Errors in diet, such as excess of alcohol (which retards digestion), ice, and many other articles which vary with the idiosyncrasy of the individual.

Prognosis and Treatment.—Acute dyspepsia of the kind here referred to usually passes off in two or three days. (1) If pain be present, assist vomiting by mild emetics, such as copious draughts of salt and water, tickling the fauces, etc. Violent emetics aggravate the condition. (2) Three grains of calomel, and milk diet for a day or two, generally relieve the condition. (3) Bismuth and tonics may be given during convalescence.

II. *The patient complains of considerable* PAIN *or discomfort, and* TENDERNESS IN THE EPIGASTRIUM, *with nausea or vomiting, all of which have come on somewhat suddenly.* The disease is probably ACUTE GASTRITIS.

§ **226. Acute or Sub-acute Gastritis** is relatively a much more serious disorder than the foregoing. It consists of a sudden derangement of digestion due to inflammation of the stomach. This condition is not so

much a catarrhal inflammation of the mucous membrane (excepting in cases of irritant poisoning) as of the glands of the stomach.

Symptoms.—(1) Pain, intense and burning, or a feeling of distension in the epigastrium, coming on directly after food, and accompanied by tenderness on pressure. (2) Vomiting, not always immediately after a meal, of undigested food, sometimes with streaks of blood. (3) Malaise, anorexia, slight pyrexia, headache, depression, and other constitutional symptoms may be present, attended sometimes by great prostration, thirst, furred and coated tongue. (4) Diarrhœa may ensue after a day or two.

The *Diagnosis* may have to be made from acute dyspepsia (§ 225), and from other causes of vomiting (§ 215).

Recovery generally takes place in about three to six days, the affection rarely lasting longer than eight or ten days. It may go on to chronic gastritis. Death rarely takes place, excepting from irritant poisoning or in cases of membranous gastritis.

Etiology.—(1) In the majority of cases simple acute gastritis is caused by errors in diet, or by decomposing (or infected) meat—*e.g.*, tinned food ; an excessive quantity of normal food will cause it. (2) Irritant poisons (*e.g.*, arsenic, antimony, phosphorus, etc.). In long-continued vomiting, without apparent cause, poisoning should be suspected, and the vomited matters examined. (3) In some cases, gout and other constitutional conditions predispose to or determine an attack. Heart, lung, and liver disease are predisposing causes.

Treatment.—The indications are : (1) To remove any irritant that may be present from the stomach. This can be done by promoting vomiting, which is specially indicated if the epigastric pain continues. The stomach may be washed out by syphonage. It may be desirable to give a purgative, such as 3 grains (0·19) of calomel (if there is vomiting ⅓ grain (0·03) doses hourly), and a seidlitz powder in the morning. Hot fomentations or a mustard leaf to the epigastrium may relieve the pain. (2) The second indication is rest to the stomach, which is gained by twelve or twenty-four hours' abstinence from food, followed by milk in small quantities. Later on, bismuth combined with opium is the best treatment. The milk diet should be supplemented only very gradually.

CHRONIC DISORDERS OF THE STOMACH

§ 227. *The patient, whose temperature is normal, complains of "* Chronic Indigestion,*"*—i.e., *pain or discomfort in some way connected with his food, which has probably come on gradually, and may have lasted a long time.* There are SIX DISORDERS, from any one of which he may be suffering, and there may be *Dilatation of the Stomach* in addition.[1]

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[1] As already stated, this classification is not very satisfactory. It is based on *tenderness* as a guiding sign. Lord Dawson (*B. M. J.*, June 1922) has proposed the following clinical grouping in which *pain* is the key.

1. Pain soon after food with tenderness and relieved by vomiting—infection somewhere, *e.g.*, teeth, tonsils, appendix or colon.

(a) **Functional diseases** of the stomach **without tenderness.**
   I. Atonic Dyspepsia.
   II. Acid or irritable Dyspepsia.
   III. Gastralgia.
(b) **Organic diseases** of the stomach **with marked local tenderness** and pain.
   IV. Peptic Ulcer.
   V. Cancer of Stomach.
   VI. Chronic Gastritis.
(c) There are also many other disorders unconnected with the stomach which may give rise to symptoms of chronic indigestion, among which the following may be mentioned : Phthisis (of which dyspepsia is often the earliest symptom), Appendicitis, Anæmia, Abdominal Tumour, Cardiac or Hepatic Disease, Renal or Uterine Disease, various Nervous Disorders, and Pancreatic Disease (rare).

GROUP A. **Chronic Dyspepsia** may be defined as deranged digestion without gross or inflammatory changes in the mucous membrane of the stomach. It may be, and often is, attended by Atony or Dilatation, § 234. It occurs in two generally accepted types.

I. ATONIC (HYPOTONIC) DYSPEPSIA (syn. Asthenic dyspepsia), the commoner form, is chronic indigestion due to diminished digestive power of the stomach. There is usually diminished secretion of gastric juice, and there may be delay in the stomach. But a hypotonic stomach may empty itself in the normal time, i.e., may have normal or at least effective peristalsis.

II. ACID or irritable DYSPEPSIA (§ 229 ; Synonym, hyperchlorhydria) is a chronic indigestion due to hypersecretion of gastric juice.

I. *The patient complains of* CHRONIC INDIGESTION, *and the epigastric pain or discomfort comes on* SOON AFTER A MEAL. The disease is probably ATONIC DYSPEPSIA.

§ 228. **Atonic Dyspepsia** is the commoner form of chronic dyspepsia. The *Symptoms* are : (1) Pain or distress, usually in the epigastrium, coming on immediately or *very shortly* after food. The pain may be in the back or shoot up to the shoulders ; or there may be no definite pain, only a feeling of weight or distension. It is unaccompanied by tenderness on pressure, a feature which distinguishes it from gastritis and other organic conditions. The pain is often relieved by eructations of wind. (2) Nausea and vomiting are not frequent. (3) The appetite is usually diminished ; it may be good but cease quickly after the beginning of the meal. Often breakfast is well taken, lunch not so well, and later meals worse. The

2. Pain two to three hours after food, relieved by food and alkalies,—ulcer, duodenal or gastric ; chronic appendicitis or colitis, overwork.   •
3. Pain constant, with distension and flatulence,—fatigue, anæmia, cancer.
Ryle (*B.M.J.*, Jan. 1923) classifies dyspepsias into the following groups according to the cause (see also the *Lancet*, April 4, 1925):
   (a) Organic disease of stomach or duodenum—ulcer, cancer, gastritis, post-operative ;
   (b) Reflex from gall-bladder, appendix or central nervous disease ;
   (c) Unphysiological life—overeating, alcohol, smoking, overwork, constipation ;
   (d) General ill-health—tuberculosis, anæmia, visceroptosis ;
   (e) Nervous tricks, emotion, worry.

tongue is flabby and indented by the teeth. (4) There are languor, depression, and general discomfort and drowsiness after meals. There may be palpitation, dyspnœa, and other cardiac symptoms. Thirst is not usual unless there be dilatation, and pyrexia is absent. Urates in excess are constantly present in the urine. Sometimes erythema faciei and urticaria occur.

Etiology.—(1) Errors of diet ; (2) overwork, mental anxiety, and other nervous derangements ; (3) imperfect mastication in previous years ; (4) convalescence from acute diseases, anæmia, and lowered vitality or debility from any cause. Dyspepsia is often the earliest symptom met with in phthisis. (5) Various abdominal disorders—*e.g.*, pancreatic or renal disease,[1] appendicitis, chronic colitis, enteroptosis, floating kidney, and abdominal tumour, may for some time be evidenced only by symptoms resembling atonic dyspepsia. (6) It may be part of organic disease of the stomach, such as syphilis or early pyloric cancer.

Diagnosis.—The chief condition from which it has to be distinguished is *chronic gastritis*, in which there is usually tenderness on pressure ; and, while stimulating articles of food (pickles, condiments, etc.), relieve the pain of atonic dyspepsia, they tend to aggravate chronic gastritis (see also Table XV.). Atonic dyspepsia may have to be differentiated from *gastric ulcer* in the young, or *cancer of the stomach* in the middle-aged and old (*q.v.*). The differentiation from *acid dyspepsia* is given in § 229, but it must be remembered that in atonic dyspepsia with delay butyric acid fermentation may take place, which is distinguishable from acid dyspepsia only by an examination of the stomach contents. *Achylia gastrica* may be attended by similar symptoms, and in addition acne rosacea, visceroptosis and ileal stasis.

Prognosis.—It is never fatal, but often renders life so wretched as to unfit the sufferer for the duties of life. If met with early, treatment may be very efficacious ; but, if untreated, it may go on to chronic gastritis and dilatation of the stomach, and lead to general malnutrition (§ 218). The results of test-meal and X-ray examinations will form the best basis for rational treatment.

Treatment.—The indications are, to remedy the dietetic errors (see § 236) ; to remove the cause ; and to stimulate the secretory and motor power of the stomach. As a rule farinaceous and sloppy foods are useless. The teeth, gums, and tonsils should be seen to. Bismuth and alkaline carbonates shortly before meals, combined with nux vomica, bitters, and carminatives (Formula 66), stimulate the secretory powers of the stomach. Some cases do best by taking their meals dry, so that the gastric juice may be undiluted. The smaller bulk of food does not dilate the stomach so much ; thus the organ can empty itself better. Some find aid in pepsin, lactopeptine, taka-diastase, or other artificial digestive. Dilute hydrochloric acid ℔xx to ʒi (1·3 to 3·9), after meals, with strychnine, suits some cases better than the alkalies before meals. Attention to the general

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[1] Appendicitis is usually associated with acid dyspepsia.

health may succeed where stomachic treatment alone fails. Abdominal massage, electricity and exercises to promote muscular contraction are important curative measures. Rest before and after meals is excellent in nervous cases. Various *symptoms* require treatment. For the flatulence 20 grains (1·3) of sodium bicarbonate in a cupful of hot water gives great relief. Peppermint, sp. chloroformi, rhubarb, ginger, cardamoms, sodium sulphocarbolate, charcoal, or Formula 50, are all useful. Acid eructations may be counteracted by antiseptics (carbolic acid, charcoal), or alkalies (cremor magnesiæ, Extra Pharmacopeia, and bismuth). Hydrocyanic acid and opium (with caution)[1] may be used for pain. If hypotony with or without poor gastric juice, daily lavage of the stomach is valuable. In constipation cases, improvement of colon activity seems to improve or cure the stomach symptoms. For breathlessness, palpitation, and other cardiac symptoms, sal volatile and alkalies may be given. Compare also Treatment of Chronic Gastritis (§ 233).

TABLE XV.

	CHRONIC DYSPEPSIA.	CHRONIC GASTRITIS.
Tenderness .	Absent.	Present.
Vomiting .	Not frequent, but relieves pain.	Frequent, especially in the morning, of mucus; no relief.
Thirst . .	Varies; not common.	Usually marked.
Fever . .	Absent.	Sometimes slight fever.
Causes . .	1. Dietetic errors. 2. General weakness of system (anæmia after fevers, etc.); or nervous exhaustion, leading to *deficient secretion* of gastric juice, or *deficient motor activity* of stomach.·	1. Dietetic errors, especially alcoholic excesses. 2. Sequel to Heart or Liver Disease.
Course . .	Liable to come on in attacks, lasting a few days or weeks at a time; brought on by slight causes.	Does not come and go, but progressively advances, and goes on to dilatation of the stomach.

II. *The patient complains of* CHRONIC INDIGESTION, *but the discomfort does* NOT COME ON SOON *after a meal, and is relieved by food.* The disease is probably ACID DYSPEPSIA.

§ 229. Acid Dyspepsia (Superacidity, Hyperchlorhydria, compare § 223) may be defined as chronic indigestion due to excessive secretion of gastric juice. It is frequently due to spasm of the pylorus, set up reflexly from abnormal conditions in other parts of the alimentary canal. Some-

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[1] De Quincey started his habit of "opium eating" for an intractable form of chronic dyspepsia.

times it is a *chronic glandular gastritis*, set up by local irritation of injudicious food or alcohol.

The *Symptoms* which distinguish this from Atonic Dyspepsia, which it resembles in other respects, are : (1) Pain, severe, gnawing, intense, burning, *coming on one or two hours after food ;* unattended by tenderness on pressure ; and usually relieved by taking food or alkalies. (2) There is usually a good or even excessive appetite ; there may be acid eructations, which may be so acrid as to make the throat sore, and thirst. (3) *The presence of HCl in an empty stomach,* say, before breakfast, is the crucial test of hypersecretion. When the secretion of acid is very excessive, attacks of vomiting of HCl occur, lasting a few days. The patient may awaken with pain in the middle of the night. This condition is named *gastro-succorhœa.*

Etiology.—(1) It is usually a reflex hypersecretion, or retention with pyloric spasm, when colitis, appendicitis, cholelithiasis, gastric or duodenal ulcer are present. In some cases it appears to be a pure neurosis. (2) It may arise from excess in alcohol, or highly spiced foods, or simply over feeding. (3) It is usually met with in healthy adults, or men in the prime of life. (4) Superacidity may be found without any symptoms, and there is some evidence that it may be familial.

Diagnosis.—For the diagnosis from *Chronic Gastritis,* see § 233, and Table XV., p. 315. *Gastralgia* may simulate acid dyspepsia ; but in the latter the pain is relieved by alkalies, while in gastralgia it is not so relieved The examination of the stomach contents shows that in hyperchlorhydria the proteins are more completely digested than in gastralgia. Many cases of *Duodenal ulcer* have a history of hyperchlorhydria preceding the more serious symptoms. The occult blood test differentiates this. *Achylia Gastrica* may simulate the acid eructations and all the gastric symptoms of acid dyspepsia, except perhaps pain. Relief even may be obtained from taking food and alkalies ; but there is undigested food in the fæces and sometimes diarrhœa.

The *Treatment* is mainly dietetic (see § 236). A diet of proteins relieves the condition, but if persisted in too long, this further stimulates the secretion of HCl. An exclusively proteid diet used always to be prescribed for cases of hyperchlorhydria ; much on the lines of the Salisbury diet. Sometimes it gives very good results. At the beginning olive oil ʒi—ʒi (4—32) is given before meals because fats have an inhibitory action on gastric secretion. The diet contains meat, eggs, fish, macaroni, rice, milk and suet puddings, cream, bacon and fatty foods. Meat extracts and highly spiced foods, wines, spirits, coffee are forbidden ; and sweets, fruits and vegetables and tobacco are to be taken in small quantities only. No fluid should be drunk during the meal, but a good quantity should be taken at the end of the meal. Antacids, such as large doses of sodium bicarbonate, heavy magnesia, or creta preparata, may be given one to two hours after meals. Lozenges constantly and slowly sucked, which induce a considerable amount of alkaline salivary secretion, are useful,

especially the bismuth, magnesia, and chalk lozenge (B.P.). Atropin, gr. $\frac{1}{150}$ (0·0004), before meals or belladonna and bromide inhibit secretion. As a temporary measure, the acidity of the stomach may be diluted by a copious draught of hot water, which relieves the pain and acid eructations. (Compare diet for duodenal ulcer, § 231.)

III. *The patient complains of sharp paroxysmal* PAIN, *having* NO DEFINITE RELATION *to the taking of food, and careful investigation reveals no structural disorder of the stomach.* This case is probably one of GASTRALGIA.

§ 230. **Gastralgia** is a gastric neuralgia, sometimes attended by a hyperæsthesia of the mucous membrane of the stomach, but always without structural changes or alteration of secretion.

Symptoms.—(1) The pain is of a sharp or burning character in the epigastrium, usually relieved by pressure. There is generally no tenderness, but if present, it is usually more marked with a light than a heavy touch, thus differing from organic disease. Sometimes it is unilateral. The pain may begin immediately after food, but may come on either when the stomach is empty or when it is full. The *irregularity of its advent* is one of its most characteristic features. Sometimes it comes on with the first mouthful of food ; sometimes food relieves it ; sometimes it occurs in attacks unrelated to food. Dieting gives no relief, for it may be worse after a milk diet than after raw apples. (2) Vomiting and other symptoms are rare. (3) It generally occurs in neurotic people, who have had neuralgia elsewhere. (4) It may accompany gastric ulcer, or follow this and other diseases of the stomach.

Etiology.—(1) Gastralgia may come on at any age, and in either sex. (2) Some constitutional state, such as hysteria, neurasthenia, anæmia, ague, alcoholism, or gout, is usually present at the same time. (3) In tabes dorsalis, gastralgia is the most frequent form of crisis (*crise gastrique*).

Diagnosis.—Those cases of gastralgia in which food relieves the pain have to be diagnosed from *Hyperchlorhydria*. The diagnosis in such cases is effected, first, by administering alkalies an hour or so after meals ; they relieve the pain of hyperchlorhydria, but not that of gastralgia. Secondly, acid eructations are a prominent feature of hyperchlorhydria, but not of gastralgia. Thirdly, excess of hydrochloric acid is found on examining the stomach contents in hyperchlorhydria. *Ulcer of the stomach* has a more limited area of tenderness on pressure ; the pain comes on immediately after food, and is relieved by vomiting. Increased HCl is found on examination of the vomited matter. *Cancer of the stomach* is very difficult to diagnose from gastralgia before tumour or hæmatemesis supervenes, but in this disease the pain is usually more constant. *Biliary colic* is usually associated with jaundice.

Treatment.—(1) Treatment directed to the constitutional condition generally relieves the gastric trouble sooner or later. (2) Warmth to the epigastrium, opium (with caution), nitrate of silver internally, gr. $\frac{1}{6}$ (0·01), and arsenic in small and frequent doses. For the vomiting give hydrocyanic acid, bromides, and liq. arsenicalis ($\mathfrak{m}$ i. (0·06) in a drachm (4) of water every half hour).

GROUP B. If the patient complains of **Chronic Indigestion,** attended by pain and marked tenderness on pressure, we are justified in suspecting the presence of organic disease of the stomach—viz., IV. SIMPLE ULCER ; V. CANCER ; or VI. CHRONIC GASTRITIS.

§ 231. **Simple or Peptic Ulcer** may be acute or chronic, and may be situated in the stomach, or the duodenum as far as the ampulla of Vater. The ulcers probably arise by peptic digestion of areas of mucous membrane which have been spoiled by toxins swallowed from the mouth or pharynx, or absorbed from septic foci elsewhere in the body. They tend to heal

readily unless there is gastric stasis and superacidity, when they become chronic, erode the wall of the viscus and may invade adjacent organs.

IV a. *The patient is an anæmic young woman, and complains of severe* PAIN, PRODUCED BY FOOD *and* RELIEVED BY VOMITING, *the vomit sometimes containing a large quantity of blood.* The disease is ACUTE ULCER OF THE STOMACH.

Acute Peptic Ulcer occurs in the second and third decades of life, and is more frequent in females than males. The ulcers are usually small and multiple. There are three very characteristic features, tô which the symptoms of chronic dyspepsia may be added :

(1) *Pain* of an intense boring character usually limited to ونe spot, (2) *aggravated by food,* and accompanied by tenderness. A small, very tender area, pressure on which even by the bed-clothes cannot be borne, is sometimes present, and is very characteristic. It is usually situated in the epigastrium. (3) The pain is *relieved by vomiting,* which comes on very shortly after food. The vomited matter contains an excess of hydrochloric acid. (4) Hæmatemesis, which may be profuse, comes on suddenly from time to time. (5) The appetite is usually normal or increased but the patient avoids food because of the pain it produces. There is generally constipation, and anæmia, and often a history of inadequate food and lack of fresh air. In some cases there may be no symptoms until profuse hæmorrhage or perforation suddenly occurs.

The *Diagnosis* is not difficult if pain, an area of tenderness, and hæmatemesis be present. The last, which was thought to be the most characteristic symptom, is now known to be very profuse in gastrostaxis (§ 216). Chronic appendicitis may simulate the disease.

Treatment of Acute Ulcer.—In all but the mildest cases the patient must rest in bed. Treatment of hæmorrhage is given in hæmatemesis. In cases of perforation immediate laparotomy is required. Where there is recent hæmorrhage or intractable vomiting, no food is allowed by the mouth but ice may be sucked. Feeding is solely per rectum. The average case of acute ulcer in young girls is usually cured by rest in bed, relief of constipation, and treatment of the anæmia with iron and arsenic. The diet in these cases should not be restricted. The only contra-indications to full diet are hæmorrhage, vomiting, perforation or chronicity. All sources of infection—teeth, tonsils, appendix, gall bladder—must be removed. Chronic inflammation of the appendix must be remembered. The Lenhartz diet is rarely required (see p. 319).

IV*b. The patient is a middle-aged man, with* RECURRING ATTACKS OF PAIN *an hour or two after food, accompanied by* TENDERNESS IN THE EPIGASTRIUM, *and* RELIEVED BY *emptying the stomach naturally, or by* VOMITING. *The disease is probably* CHRONIC ULCER OF THE STOMACH.   •

Chronic Ulcer of the Stomach is usually single, and occurs more frequently in males and between the ages of 30 and 50. The patient is thin and miserable, and complains of (1) " chronic dyspepsia." (2) Attacks

of pain in the epigastrium, left upper abdomen or back, come on one or two hours after a meal and pass off before the next meal. (3) The appetite is restrained, as he is afraid to take solid meals, and feels better when resting and taking light food. (4) Vomiting is not frequent, but is sometimes induced by the patient in order to get relief, when the fluid is acid and contains well-digested food. (5) Hæmatemesis is not frequent. Constipation is usual.

The *diagnosis* is not difficult if the characteristic pain and tenderness are present, but pain after food, relieved by emptying the stomach, with occasional vomiting of blood, may occur in other diseases. Pain coming on one-and-a-half to two hours after food, and passing off before the next meal, is typical of gastric ulcer, and occurs unless there are complications of the local condition (such as adhesions to surrounding organs or chronic perforation) or accompanying disease elsewhere in the abdomen (appendix, gall-bladder). Every case of " chronic dyspepsia " should be investigated. The test-meal and efficient X-ray examination are the only means of coming to a diagnosis. The stomach contents, withdrawn after a test-meal, show increased total acidity (50–100) and active chlorides (0·20–0·30). In the X-ray examination the cavity of the ulcer projecting into the wall of the stomach may be filled with the opaque material, or a spasm of the circular muscle of the stomach opposite the ulcer may be seen. But a single upright photograph may not reveal the ulcer ; it is necessary to get photographs of the curvature in profile, at different angles, and to observe the arrest of peristalsis at the ulcer.

Prognosis.—The prognosis is usually favourable if the condition is treated early, but there is a great tendency to relapse. If untreated, perforation into the peritoneal cavity may cause death (see § 193). When a more favourable course is followed, the resulting cicatrisation may lead to distortion or stricture of the stomach or pylorus. Stricture of the pylorus leads to dilatation of the stomach. Stricture of the stomach leads to the " hour-glass " contraction—*i.e.*, the stomach is divided into a cardiac and a pyloric cavity. Adhesions to surrounding viscera, sub-phrenic abscesses, or abscess in other situations may result. Death occasionally results from hæmorrhage. The amount of the bleeding is no measure of the size or depth of the ulcer.

Treatment of Chronic Ulcer.—The remarks on the treatment of acute ulcer during the acute stage, when hæmorrhage of vomiting is present, are true here also (p. 318). Formerly, after the acute stage a gradual return was made to ordinary diet, beginning with citrated or peptonised milk, then custard and bread and milk, then bread and butter, eggs, fish, and chicken. Lenhartz introduced a modification of this treatment based on the theory that the subnutrition induced by starvation and rectal feeding was prejudicial to the healing of the ulcer, and that gastric juice in a fasting stomach was irritating to the ulcer. The food given must be such as will neutralise the stomach acid, will excite little secretion, and, thirdly, of a bulk which will not distend the organ. Absolute rest in bed

is essential for four weeks, or longer ; and getting up must be very gradual. The diet consists of raw eggs beaten up with sugar, and iced, and of milk, taken in small quantities frequently during the day, combined with olive oil when this can be taken. On the first day one egg and 7 to 10 ounces of milk are given. Every day one egg and 3½ ounces of milk are added till eight eggs are taken daily. Raw minced meat (1 ounce) is added about the sixth day, later if ulceration still active ; then boiled rice and soft bread. Gradually meat and pounded fish are substituted for the eggs, and by the end of the fourth week light ordinary food is taken,

TABLE XVI

	CHRONIC ULCER.	MALIGNANT DISEASE.	CHRONIC GASTRITIS.
Pain . . .	1½–2 hours after food.	Constant discomfort.	Immediately after food.
Vomiting .	Not frequent ; relieves pain.	Often large quantity every few days.	Morning vomiting of mucus.
Hæmatemesis	Occasional but profuse ; therefore bright red.	A continuous oozing ; therefore "coffee-ground" in character.	Rare ; and only streaks, unless in the venous congestion due to heart disease.
Tumour . .	None.	Present, though may not be palpable ; secondary deposits may be recognisable in liver, peritoneum, glands, etc., later on.	None.
Age . . .	Usually men, thirty to fifty.	Usually men over forty.	Any age.
Course . .	Indefinite ; relapses occur.	Fatal in one to two years if not removed.	Indefinite ; may go on to dilatation.

but a careful after diet should continue for three months. Fat, oil and cream restrict the acid gastric secretion. Rectal salines relieve thirst. Large doses of bismuth are given at the beginning. No aperients are taken during the first week, the bowels must be kept open by an enema every second day.

In the intervals between the acute attacks alkaline carbonates, magnesia, bismuth, and many of the remedies used in gastritis (q.v.), have been recommended. For the pain, give hydrocyanic acid, opium, bismuth, and alkalies. In very chronic cases nitrate of silver may be tried. To regulate the bowels, the best form of aperient, if enemata fail, is a drachm

(4) of Carlsbad salts in 3 (96) or 4 (128) ounces of water (120° F.) taken every fifteen minutes in four doses up to half an hour before breakfast.

The indications for operation in gastric ulcer are (i.) perforation; (ii.) for frequently recurring obstinate cases; (iii.) for repeated hæmatemesis; and (iv.) pyloric obstruction and hour-glass constriction. For perforation, immediate operation is imperative. For (iii.) surgical measures should not be lightly employed, since recurring and severe hæmatemesis may be unassociated with ulceration (see Gastrostaxis, § 216). Moreover, recurrence is by no means uncommon, after excision of the ulcer or gastro-enterostomy has been performed. If, in spite of adequate treatment, such as is detailed above, the pain, vomiting, or bleeding prove intractable, operation must be considered.

IVc. *The patient is a healthy looking, active, middle-aged man, who for years has had attacks of acidity after overwork, worry or indigestible food, who has* PAIN *coming on three to four hours after food or in the night,* RELIEVED BY TAKING FOOD. *The disease is* DUODENAL ULCER.

Duodenal ulcer is four times as frequent in males as in females. The symptoms begin at the age of 20–35, and tend to come in attacks after dietetic errors, overwork, worry or exposure. (1) Epigastric pain, sometimes intense, comes on when the stomach is empty, three to four hours after food, the so-called " hunger pain," which frequently wakes up the patient about 2 a.m., and is relieved by food. The pain tends to come at a regular time each day and during the night. (2) Tenderness may be present just above, and to the right of, the umbilicus. (3) The pain may radiate to the back and become more constant if the ulcer burrows into the head of the pancreas. (4) There may be sudden intestinal hæmorrhage, evidenced by melæna, preceded or accompanied by hæmatemesis. (5) Hyperchlorhydria is usual. (6) X-ray examination reveals either pyloric spasm and delay, or the contents rush through and there is rapid emptying of the stomach. A series of radiograms, rapidly taken, may reveal characteristic irregularity of the duodenal cap.

Diagnosis.—The characteristic pain makes the diagnosis easy in typical cases of duodenal ulcer. Chronic gastric ulcer, stone in the gallbladder or kidney, and chronic appendicitis are to be differentiated. The diagnosis is confirmed best with the X-rays. A fractional test-meal and the discovery of occult blood in the stomach and fæces may help in obscure cases. Similar symptoms, but with left-sided pain, may accompany ulceration occurring after gastro-enterostomy.

Prognosis.—Medical treatment is usually successful, but it must be adequate. Insufficient treatment is the cause of non-success, as the ulcer readily heals superficially, but tends to relapse easily unless time is given to allow healing to take place throughout. Perforation, hæmorrhage or recurrence after thorough medical treatment are indications for surgical interference. Operation should always be followed by careful medical treatment.

Treatment.—The indications for treatment are (1) to rest the patient,

O.M.

(2) to reduce the superacidity of the gastric juice, and (3) to eliminate all sources of sepsis. All foci of infection, e.g. septic teeth, infected tonsils or chronic appendix, must be treated; and it should be stipulated that the patient be in bed for four to six weeks. The diet consists at first of small quantities of olive oil every two hours. Milk or cream and egg are added after a day or two. Thirst may be relieved by sips of alkaline water (bicarbonate of soda) a teaspoonful to a pint, and by saline enemata. The quantities of egg and milk are increased slowly, and the intervals between feeds lengthened. Well boiled rice, minced chicken, rusks and ' butter, milk puddings are gradually added, and at the end of three or four weeks a light invalid diet is reached. All mechanical and chemical irritants should be avoided in the diet. The bowels may be kept open by a glycerine suppository or an enema every second day. Full doses of bismuth, or cream of magnesia are useful at the beginning, half an hour after each meal; or teaspoonful doses of a powder containing bismuth carbonate gr. x. (0·6 gm.) bicarbonate of soda gr. xx. (1·2 gm.), magnesium carbonate and calcium carbonate āā gr. x. (0·6 gm.). Tr. belladonna in 5 drop doses relaxes spasm, and with olive oil, inhibits secretion. Sippey recommends that in the early part of the treatment, in order to inhibit secretion, Tr. belladonna in 5 drop doses should be given alternately with a tablespoonful of olive oil, before each feed. The patient is allowed up very gradually. For at least three months only light work should be done. The treatment of hæmorrhage has been given above (§ 216).

V. *The patient, who is in middle or advanced life, presents more* CACHEXIA *than could be accounted for by dyspepsia, and vomits from time to time* "COFEE-GROUND" MATERIAL. There is probable MALIGNANT DISEASE OF THE STOMACH. Gastric symptoms beginning in a patient of middle age or over should always be regarded as suspicious of cancer.

§ 232. Cancer of the Stomach.—The stomach is a frequent site for primary cancer; it has been found in as many as 1 per cent. of all post-mortems. The disease may attack any part of the stomach. The word " cancer " is associated in our minds with a tumour, but in two-thirds of the cases of cancer of the stomach there is no tumour, but a scirrhus infiltration of the pylorus, which produces obstruction of that orifice and leads to Dilatation (§ 234). The clinical history, which rarely extends beyond one or two years, may be described in three stages. In the first stage we find the symptoms of chronic gastritis (§ 233), combined with marked cachexia. In the second stage, combined with these are acute pain (generally), vomiting, and hæmatemesis of a very characteristic kind. In the third stage, besides the preceding, we get either dilatation of the stomach, or tumour, or both. In many cases, however, there are no symptoms referable to the stomach, and the diagnosis is only made in the deadhouse.

Symptoms.—(1) *Loss of appetite,* soon followed by *cachexia,* occurs early, and is very marked; and these symptoms in a patient of 40 or

upwards should always make us suspect the condition. The sallowness of the skin may almost pardonably be mistaken for pernicious anæmia, or even jaundice. (2) The *pain* is situated in the epigastric region or back, radiates in different directions, and is usually accompanied by tenderness. It is continuous, sometimes increased by food, but sometimes independent of the taking of food. (3) *Vomiting* is a fairly constant sign. Generally it takes place some time after the ingestion of food, the interval depending upon the position of the lesion ; thus, if at the cardiac end, the interval is short ; if at the pylorus, it may be hours after taking food. Sometimes the vomiting occurs every two or three days. An examination of the vomited matter shows diminution or absence of hydrochloric acid and the presence of lactic acid. (4) *Hæmatemesis* is generally present sooner or later. The bleeding is small in quantity, but occurs frequently, and therefore the blood is partly digested, and gives rise to a characteristic brown appearance, as of *coffee-grounds*. Occult blood is found in the stools. Persistent bleeding occurs if a vessel is eroded by the growth. (5) *Dilatation* of the stomach is sure to ensue if the pylorus is involved (§ 234), Sarcinæ and other evidences of decomposition may be present, and sometimes cancer cells. (6) *Tumour* is much less rarely met with than one would expect. Transmitted aortic pulsation, and a little fulness or rigidity of the upper end of the right rectus, may be present without a palpable tumour. When cancer is deposited in the pylorus, it may cause adhesions which prevent the tumour from coming forward. The great majority of gastric tumours come forward to the left of the middle line. It is usually stated that whereas hepatic tumours move, gastric tumours usually do not move with respiration ; but this feature has many exceptions. One of greater importance is their alternate appearance and disappearance. At first they are extremely mobile, but later on they become fixed owing to adhesions. This is also the reason why perforation is rare. Pressure elicits pain. Distension of the stomach by copious draughts of water may help us in the physical examination.

Etiology.—(1) Cancer of the stomach is more frequent in men. (2) It is rarely met with under forty, although I have seen one case of twenty-eight, another of thirty, and several between thirty and forty years. (3) Simple ulcer and chronic gastritis appear to predispose.

Diagnosis.—Anorexia and cachexia are the only constant symptoms. The diagnosis is made best by radiology. There is a characteristic irregular outline of the stomach wall, and defective or absent peristalsis. With cancer of the body, there is a tube-like stomach with food rushing through ; in pyloric cancer there is obstruction and dilatation. The chemical examination of a test-meal is of great value. The total acidity is low, free hydrochloric acid is absent, active chlorides are present only in small amount, blood is often present, and organic acids may be found where there is dilatation. If emaciation be rapid, and gastric symptoms resist treatment, cancer should be strongly suspected. *Dyspepsia* and *chronic gastritis* have pain directly related to food ; for these, and *Simple*

ulcer of the stomach, see Table XVI., p. 320. For *Simple* pyloric stricture, see Dilatation. *Tumour of the pylorus* or stomach has to be diagnosed from tumour in the neighbouring regions (§ 212). Thus a growth on the back of the stomach may resemble a kidney tumour. *Addison's disease* and other cachectic conditions must be excluded (Chapter XVI). *Pernicious anœmia* is sometimes strongly suggested by the colour of the patient but in this disease there is not a corresponding amount of emaciation, and the blood-picture is different.

The *Prognosis* is very grave. The duration is rarely longer than six' to eighteen months after the first definite symptoms appear. Death is the invariable result unless surgical measures are adopted early. The symptoms upon which one relies most in the diagnosis in these cases, anorexia and emaciation, have always appeared to me to be those which also best measure the longevity of the patient. Death generally takes place by inanition, but almost as often it occurs suddenly by the involvement of important structures, and it would be unwise to assume that because the patient does not waste he will not die soon. Partial gastrectomy is successful if undertaken early. A clinical diagnosis without X-rays and test-meal is almost never early enough ; the chance for successful treatment lies in the early investigation of cases of dyspepsia.

Treatment.—Early surgical treatment affords most hope of success. Apart from this the indications are to support the strength and relieve the symptoms. The former may be accomplished by easily digestible or predigested food (§ 236), and by the use of pepsin and hydrochloric acid. For the latter consult § 234, Dilatation. For the flatulence and pain, creosote and opium, or morphia hypodermically. X-rays diminish the pain, and in intensive doses retard, even arrest, the growth of the neoplasm. Radium also can be tried.

VI. *In addition to other symptoms of* CHRONIC INDIGESTION, *the patient who has been, perhaps, the subject of chronic alcoholism, or cardio-pulmonary disease*—VOMITS MUCOUS IN THE MORNING, *sometimes streaked with blood.* The disease is probably CHRONIC GASTRITIS.

§ 233. **Chronic Gastritis** may be defined as a form of chronic indigestion due to parenchymatous inflammation (*i.e.*, chiefly of the glands) of the stomach.

Symptoms.—(1) Pain coming on shortly after food, usually of a dull character, and attended by tenderness on pressure. (2) Mucous vomiting in the morning, or, indeed, mucus found in the stomach contents at any time, is a very characteristic feature of chronic gastritis. • Streaks of blood are occasionally present. (3) Thirst is also a prominent feature. (4) A slight degree of pyrexia is sometimes present. (5) The appetite is usually good, but the first few mouthfuls of food satisfy. (6) Flatulence, and other symptoms, as in *atonic dyspepsia* (§ 228). (7) General symptoms are invariably present—depression, nervousness, anæmia, loss of flesh, sallowness, and other symptoms referable to the causes of the condition (see below). Chronic gastritis may constitute an early phase of cancer

—a fact which it is well to remember; the loss of appetite is then very marked.

Diagnosis.—Atonic dyspepsia, which has no tenderness on pressure, and no mucous vomiting in the morning, and *cancer* are differentiated in Table XVI., p. 320.

Etiology.—(1) Persistent dietetic errors, especially *alcoholic excesses.* (2) Venous congestion, arising either from cirrhosis of the liver, or from heart disease. (3) It may be a sequence of repeated attacks of acute gastritis. (4) Constitutional debility, such as that in Bright's disease, gout, etc., may predispose; and so also may (5) Local causes, such as cancer, ulcer, and stricture of the pylorus.

The *Prognosis* depends a good deal on the cause and the duration of the symptoms. The case is more grave when due to irremovable venous obstruction. If the disease remain long untreated, the stomach becomes dilated, the walls fibrous, and the glands impaired or destroyed. There are three stages : First, simple *congestion*, in which the hydrochloric acid is diminished, and lactic and fatty acids are found. The second stage is one of *mucous catarrh*, in which there is a large secretion of mucus, hydrochloric acid is almost completely absent, and very little pepsin is present. In the third stage there is *atrophy* of the mucous membrane. In this stage both hydrochloric acid and pepsin are absent. Simple alcoholic gastritis soon does well.

Treatment.—(1) Here again a correct diet is the most important indication (§ 236). Give small quantities of *dry* food at long intervals (six or more hours). Alcohol and condiments should be stopped, and smoking must be interdicted. (2) The medicinal indications in the first stage and in mild cases are (i.) to promote the flow of gastric juice and stimulate the stomach power by bitters, gentian, quassia, nux vomica, and carminatives ; (ii.) stimulate the secretion of the stomach by alkalies and bitters given before meals, or aid the defective secretion by giving hydrochloric acid after meals. (3) Symptomatic treatment : For the pain, bismuth, magnesium carbonate, and opium ; for fermentation and acidity, alkalies, two or three hours after a meal. Mucous vomiting is relieved by draughts of hot water, with alkalies, before breakfast. If the appetite is too keen, give bismuth and magnesium carbonate ; in this condition bitters are harmful, as they excite the nerve-endings in the stomach. In the later stages the indications are (i.) to replace the absent gastric secretion, which is done by giving pepsin, papain, hydrochloric acid, and predigested foods ; (ii.) to prevent fermentation, give alkalies and antiseptics some time after meals, such as creosote, charcoal, carbolic acid, and sulphocarbolate of sodium.

GROUP C.—*The patient presents symptoms of* CHRONIC INDIGESTION, *and on physical examination there is* SPLASHING, *or the* AREA OF THE STOMACH RESONANCE *is increased, or there are* FOOD RESIDUES *before breakfast.* The disease is probably GASTRIC ATONY or DILATATION.

§ 234. Gastric Atony and Dilatation of the Stomach are conditions which may accompany or succeed many of the preceding disorders. Gastric Atony, the importance of which has been previously referred to (§ 222) is insufficiency of the power of the stomach to empty itself, independently of pyloric obstruction.

(a) GASTRIC HYPOTONY (Motor Insufficiency) may, it appears, exist in three stages or degrees   (a) In simple *loss of tonicity* the stomach is able to empty itself, but there is delay, and splashing can be elicited during, the period of digestion, which is prolonged. Many of these cases are latent, and exhibit no symptoms for a considerable time.   (β) *Stagnation myasthenia gastrica*, where the stomach cannot empty itself before the next meal, though it does so during the night.   (γ) *Retention myasthenia gastrica*, or true dilatation, in which the stomach cannot empty itself during the night, and at all times contains food residues, even when examined by the tube before breakfast. The symptoms of gastric hypotony are (1) prolonged lassitude after meals, with other symptoms of delayed digestion and atonic or irritable dyspepsia (§ 228) ; (2) " splashing " several hours after a meal (§ 221) ; and (3) on percussion some hours after a meal there will be an enlarged area of resonance, particularly to the left of the middle line. This test may be aided by the patient taking a draught of some aérated water, or a solution of sodium bicarbonate, 53 grains (3·5 gm), followed by tartaric acid, 45 grains (3 gm), in solution (which generates at the body temperature just 1 litre of CO_2), or by inflation of the stomach by a suitable apparatus.   (4) The method mentioned in § 222, with the opaque meal and X-ray examination, affords a ready means of detecting and measuring gastric atony.

(b) GASTRIC DILATATION may be a consequence of gastric atony, or due to pyloric obstruction. Its symptoms are (1) the same as those of gastric atony in a more marked degree ; and (2) definite food residues found in the stomach before breakfast, without which one would not be justified in believing that a condition of permanent dilatation existed In all cases of suspected dilatation the stomach should be examined by the tube in the early morning, after a long fast. This also gives an important clue to the substances in which digestion is defective.   (3) A swelling may be seen below the umbilicus. Visible peristaltic movements in the epigastric region may occur when the dilatation is due to pyloric obstruction. They may be started by palpating or sharply flicking the abdominal wall.   (4) One of the most characteristic symptoms of dilated stomach due to pyloric obstruction is the vomiting, at intervals of two or three days or more, of large quantities of acid frothy material, containing sarcinæ, on which a scum forms on standing. Vomiting may be altogether absent, but if it is present and has these characteristics we may be satisfied that there is dilatation.   (5) The remaining symptoms vary with the *cause*, of which there will be a history, or evidence at the time (*infra*).   (6) Autotoxic symptoms invariably ensue—marked lassitude, and various other functional nerve symptoms ; sometimes urticaria

and other eruptions. Tetany is one of the sequelæ in severe cases.

Etiology.—Dilatation of the stomach may be a consequence of one of two conditions—ATONY OF THE MUSCULAR TISSUE (*a* and *g* below), or PYLORIC OBSTRUCTION.

(*a*) Gastric Atony may occur after prolonged overfeeding, " bolting " the food in early life, alcoholism, chronic dyspepsia (and its causes), or chronic gastritis. Rheumatism, enteric, influenza, and other acute infections have also been mentioned ; and there is no doubt that states of general debility and anæmia, such as are associated with phthisis (especially when combined with excessive feeding) and neurasthenic conditions markedly predispose.

(*b*) Obstruction due to a growth of scirrhus cancer at the pylorus is one of the commonest causes, and it may produce the most pronounced dilatation (§ 232).

(*c*) Pyloric obstruction may also occur from the cicatrisation of a simple ulcer of the stomach. The age and sex of the patient and the previous history are significant (§ 231).

(*d*) Pyloric obstruction may be due to pressure from without—*e.g.*, enlarged glands in the fissure of the liver, etc.

(*e*) Pyloric obstruction due to a band of adhesion is rare, and difficult to diagnose. It can only be recognised by the exclusion of other causes, and the history of inflammation of the peritoneum.

(*f*) Congenital hypertrophic stenosis, see § 215.

(*g*) Acute dilatation of the stomach is a rare variety that is often difficult to recognise. It may come on more or less suddenly in early life, or in states of general weakness and toxæmia, as in pneumonia, or after operation, with symptoms of collapse, resembling intestinal obstruction. It is a serious condition.

The *Diagnosis* of a markedly dilated stomach is not difficult ; the chief question is as to its cause. But the diagnosis of simple atony or myasthenia is always problematical unless the stomach tube or chemical tests be employed. Gastroptosis (§ 235) and Hour-glass stomach are readily differentiated by X-ray examination. In the latter, after washing out the stomach, some of the fluid is lost, and may reappear on a second washing. The malady is always troublesome. Even in atonic dilatation the cure is very tedious, but the prognosis is ultimately good if the disease be diagnosed early, and the cause removable. Malignant stricture is the commonest cause of pyloric obstruction, and unless dealt with surgically is fatal.

Treatment.—The indications are : (1) To keep the stomach as empty as possible. This may be done by diet No. IV., § 236, and by washing out the stomach.[1] It should be done last thing every night. It is best to use plain water with a little bicarbonate of soda to dissolve any mucus present. Give concentrated or predigested foods with very little fluid. Give few carbohydrates, and never at the same meal as animal foods.

[1] Method, see § 223. Sometimes Turck's double tube is used, the efferent being wider than the afferent tube, to prevent overdistension.

Carlsbad salts carry off much of the residue lying in the stomach when taken every half-hour in the early morning until purging ensues (F. 46 or 51). (2) Give tone to the muscular wall by electricity and massage. (3) Promote digestion (*vide* Chronic Dyspepsia). (4) To prevent fermentation, the symptoms of which are very troublesome, carbolic acid (1 to 3 minims (0·06–0·19)), thymol (5 grains (0·3)) or sodium sulphocarbolate (20 grains (1·2)), given preferably in a tumbler of water between meals. After lavage, creosote or calomel (½ grain (0·01) t. d.) may be given with. advantage. Surgical treatment may be needed, in cases due to pyloric obstruction, and pylorectomy and gastro-enterostomy have been successfully performed. •

Electricity is of great use in dilated stomach, not only for giving tone to the muscular wall, but also for promoting digestion and general nutrition. Faradism may be used, preferably with Einhorn's intragastric electrode, but the author has obtained very good results by means of galvanism applied externally. •

§ 235. **Gastroptosis** (Dropping of the Stomach : Enteroptosis) is a condition in which the stomach has dropped from its position. The symptoms and signs are apt to be confused with Gastric Dilatation. The lesser curvature may be obvious on inspection or palpation, but it is clearly detected by the method of inflation, or by X-rays after a bismuth meal. Intestinal stasis is usually also present, and hence an aggravated state of neurasthenia[1] is frequently associated with the condition.

Dietaries and Invalid Foods

§ 236. Less food is required in old age than in youth, and with a sedentary life than with an active or outdoor one. For a person in health three meals a day are usually sufficient ; but when a man is unable, from illness, to take more than a very small quantity at a time, he may require to take more frequent meals. Dietetic errors are a fruitful source of dyspepsia and gastritis. Too frequent meals, habitual over-feeding, and irregularity of the meals will in time derange any stomach. Deficiency of food, and long restriction to the same kind of food, induce dyspepsia by affording no stimulus to excite the secretions ; and in this connection it is well to remember that a frequent cause of failure on the part of the physician to cure dyspepsia is his disregard of this latter fact. Carbo-hydrates, especially potatoes and new bread, are particularly harmful for atonic flatulent dyspepsia. In anæmic cases with atonic dyspepsia starchy foods do not afford sufficient stimulus for the gastric functions ; proteids such as tender and underdone meat are more readily digested. It is, often a good rule to start treatment by cutting down the amount rather than by entirely prohibiting the use of certain articles of diet. Too frequent a use of condiments, spices, and tea, and of alcohol especially, lead to chronic gastritis ; while dyspepsia is induced by imperfect mastication, bolting of meals, too much fluid with meals, hard mental or physical

[1] The terms " gastric neurasthenia " and " dyspeptic neurasthenia " should be reserved for Neurasthenia of Gastric origin, as explained in the author's Clinical Lectures on Neurasthenia, fourth edition.

work immediately after eating, too cold or too hot food, or food which is badly prepared. Excess of tobacco-smoking and constipation are certainly causes of dyspepsia. Greasy and fried foods are bad in dyspepsia, because the gastric juice cannot penetrate the coating of fat. "Well-made " pastry and other so-called rich carbohydrate foods are a source of dyspepsia, especially when taken at the same meal as protein food. Hyperchlorhydria is said to be induced by protein overfeeding.

* Without appropriate dietetic rules our best efforts may fail, especially in the treatment of gastro-intestinal disorders, and other diseases which depend on the proper elaboration and assimilation of food. A few specimen dietaries will therefore be given, culled from various authors, or my own experience. These will serve as a basis for any number of other dietaries.

I. The following table is given as a guide to aid in the drawing up of a diet for mild cases of a tonic dyspepsia or chronic gastritis : *Breakfast*—Boiled sole, whiting, or flounder ; or a slice of crisp fried bacon or a soft-boiled egg ; a slice of dry toast with a little butter, or of bread (not new) and butter. *Beverage.*—One cup of cocoa or of milk and water, sipped after eating. *Luncheon.*—Chicken or game, with bread, and a little tender, well-boiled vegetable, such as spinach, vegetable marrow, or young French beans. *Beverage.*—Half a tumbler of water sipped *after* eating. *Afternoon Tea.*—A cup of cocoa or of weak tea with milk, and a slice of brown bread and butter. *Dinner* (two courses only).—Fish of the kinds allowed for breakfast, without potatoes. For sweets and dessert, a plain biscuit will suffice. Or a slice of any tender meat, such as saddle or loin of mutton, or the thick part of an underdone chop with crumbled stale bread ; custard, junket or jelly, or a little well-stewed fruit. *Beverage.* —Half a tumbler of water, with from one to two tablespoonfuls of spirit if desired.

Condiments and stimulants are good in atonic dyspepsia, but must be avoided in chronic gastritis, as tending to cause further irritation of the mucous membrane. The patient should abstain from salted and cured meats, tinned foods, sweets, pastry, raw vegetables, cheese.

II. In **Hyperacidity** game, vinegar, fruit and jam, condiments and alcohol should be avoided. The food should consist of soft, well cooked, finely cut up or minced meat, or poultry, eggs and cream cheese ; fats, oil if it can be taken, plain butter not made into sauces, icecream ; mashed potato, rice, macaroni ; weak tea—not coffee—or plain water, preferably after meals. Weak meat soups may be allowed. Milk is often difficult. If there is delay in the stomach, meals should be taken dry. Sometimes it is necessary to give food more frequently than three times a day. The patient should not sit at an ordinary table with others eating appetising food. *Early Morning.*—Weak tea without sugar or tumbler of hot water. *Breakfast.*—Eggs, boiled, poached, fried or scrambled, fat ham or bacon, crisp toast, plenty of unsalted butter and a cup of weak tea after the food is taken. *Lunch.*—Fish or well cooked meat which may be minced, little mashed potato, rice or macaroni, suet or baked castle pudding, tumbler of water after food. *Tea.*—Cup of weak tea with milk and no sugar, little bread and butter or rusk and butter. *Dinner* as lunch. Half to one ounce of olive oil half an hour before lunch and dinner, if it can be taken.

III. **Diet for Constipation.**—The first thing in the morning drink a tumbler of plain water, hot or cold, or eat an apple, pear, bunch of grapes, bananas, orange, etc. *Breakfast.*—Coffee, not too strong, with a little milk ; brown or wholemeal bread with plenty of butter, honey or treacle ; or well cooked oatmeal with cream or treacle. *Lunch.*—Sardines or olives in oil ; fish, chicken or roast meat ; vegetables, greens and salad ; cream cheese, wholemeal bread and butter ; fresh or stewed fruit with cream. *Tea.*—Coffee and milk, wholemeal bread, butter. *Dinner.*—Vegetable soup, fish or egg dish, vegetables and salads ; suet pudding ; fruit, wholemeal bread and butter. Fluids may be taken freely with meals, and half to one ounce of liquid paraffin night and morning.

IV. The "**Salisbury**" diet consists of nitrogenous food only, the meals being taken almost without fluid, but a quantity of hot water being taken between meals. The solid food administered is in a highly concentrated form, and gives the stomach a considerable rest. There is the elimination of the farinaceous and bulky substances which readily decompose and produce flatulence and kindred troubles. By reason of the dryness and small bulk of the food, a dilated or atonic stomach is enabled to resume its normal dimensions. The details of the diet are comparatively simple. One pound (1 lb.) of lean butcher's meat, chopped or scraped very fine, and so as to rid it of its white fibrous tissue, and lightly cooked, is taken per diem, divided into four or more meals. Occasionally a little well-toasted or twice baked (Zwieback) bread is allowed also. For a change, ½ pound of fish may be substituted for an equal quantity of meat. The meals are taken quite dry, or 2 ounces of fluid only ; but two hours later ½ to 2 pints of hot water are sipped. •

V. **Diet for Obesity** (§ 18) —*Breakfast* —Fish, bacon, beef, or mutton (6 ounces) ; one breakfastcupful of tea or coffee without milk or sugar, and one small hard biscuit, or one ounce of dry toast. *Dinner.*—Fresh white fish, beef, mutton, lamb, game, or poultry (6 ounces) ; green vegetables ; one slice of dry toast ; cooked fruit sweetened with saxin. *Tea.*—A cup of tea without milk or sugar ; a biscuit or a rusk ; 2 or 3 ounces of cooked fruit. *Supper.*—Meat or fish (about 3 ounces) with toast. If desired, a glass or two of sherry or claret may be taken. Fluids should be taken only at the end of meals. A cup of bovril may be taken as a stimulant.

VI. **Diet in Chronic Nephritis** (Sir Andrew Clark, modified). Tea and coffee being diuretics, must be taken in strict moderation. *Breakfast.*—A plate of oatmeal, whole wheaten meal, hominy or porridge, with cream or good milk ; bread or toast and butter ; cocoa, with plenty of milk added. Or a slice of well-cooked bacon, fish, or fat ham, may take the place of porridge. *Luncheon.*—A little fish, with some melted butter, mashed potato, and green vegetable, biscuit or bread and butter. Or a basin of vegetable soup, a bit of cheese, bread, butter, and salad. Or a milk pudding, with stewed fruit and cream, bread and butter. *Afternoon Tea.*—A cup of tea with milk, a slice of thin bread and butter, or rusk. *Dinner.*—Soup, purée of potato, chicken or rabbit, mashed potato, green vegetables, plain or milk pudding, with stewed fruit. Or boiled fish, butter sauce, a plain entrée with vegetables, milk pudding or shape, stewed fruit or blanc-mange, biscuit or bread and butter, a glass of plain or aerated water. Or fish, soup, game, or poultry, mashed potato, green vegetables macaroni cheese. *Dessert.*—Ripe fruit. *Beverage.*—A glass of plain or aerated water. *The last thing at night.*—A glass of milk and soda-water. Salt is to be carefully avoided.

VII. **Dietary for Diabetes Mellitus** (Hill & Eckmann). (1) *Semi-starvation Days.* Sample diets.

Breakfast --String beans and lettuce, āā 25 gms. ; coffee. *Dinner.*—Lettuce and cucumber, āā 25 gms. ; tea. *Supper.*—Lettuce and tomato, āā 25 gms. ; tea. Protein, 2 gms. ; fat, a trace ; carbohydrate, 5 gms ; calories, 30.

Breakfast —1 egg ; lettuce and cucumber, āā 25 gms. ; coffee. *Dinner.*—1 egg ; lettuce and string beans, āā 25 gms. ; tea. *Supper.*—Lettuce and string beans, āā 25 gms. ; tea. Protein, 15 gms. , fat, 12 gms. ; carbohydrate, 4 gms ; calories, 189.

(2) *Transition Days.* Sample diet.

Breakfast.—1 egg ; string beans, 100 gms. ; coffee ; cream, 1 oz. *Dinner.*—Chicken broth, 6 oz. ; 1 egg ; celery, 100 gms. ; tea. *Supper.*—1 egg ; 2 egg-whites ; lettuce, 75 gms. ; cucumbers, 50 gms. Protein, 36 gms. ; fat, 30 gms. ; carbohydrate, 11 gms.; calories, 471.

(3) *Full Diet Days.*

Breakfast.—1 egg ; string beans, 100 gms. ; asparagus, 100 gms. ; coffee ; cream, ½ oz. *Dinner.*—Chicken, 75 gms. ; cauliflower, 100 gms. ; olives, 25 gms. ; cucumbers, 50 gms. ; tea ; cream, ½ oz. *Supper.*—1 egg ; spinach, 100 gms. ; celery and lettuce, āā 50 gms. ; tea ; cream, ½ oz. Protein, 46 gms. ; fat, 51 gms. ; carbohydrate, 19 gms. ; calories, 740.

Breakfast.—Bacon, 50 gms. ; asparagus and spinach, āā 100 gms. ; butter ; cream, 1½ oz. *Dinner.*—Beef broth, 8 oz. ; chicken, 75 gms. ; cabbage, 100 gms. ; cucumber, 50 gms. ; butter ; icecream, 2 oz. *Supper.*—1 egg ;½ tomato, 100 gms. ; spinach, 50 gms. ; butter ; cream, ½ oz. Protein, 49 gms. ; fat, 123 gms. ; carbohydrate, 19 gms. ; calories, 1422.

And see modern text-books, such as that of G. Graham.

JOSLIN'S TABLE OF CARBOHYDRATE PERCENTAGE (*Boston Med. and Surg. Journal*).

Carbohydrates percentage : 5 *per cent. and less* in lettuce, spinach, sauerkraut, string beans, celery, asparagus, cucumbers, Brussels sprouts, sorrels, endive, ripe olives, grape fruit, unsweetened and unspiced pickle, cauliflower, tomatoes, rhubarb, egg plant, leeks, beet greens, watercress, butternuts, scallops, fish roe. 6 *per cent. or less* in : Cabbage, radishes, pumpkin, kohl-rahi, oysters, liver. 10 *per cent. or less* in : Onions, spinach, turnip, carrot, okra, beets, mushrooms, lemon, orange, cranberry, strawberry, blackberry, gooseberry, peach, pine-apple, water-melon, musk melon, Brazil nut. 15 *per cent. or less* in : Green peas, artichoke, parsnip, apple, pear, apricot, cherry, currants, raspberry, huckleberry, canned Lima beans, peccans, filberts, walnuts, pistachios, beech'nuts. 20 *per cent. or less* in : Potato, shell bean, baked bean, green corn, boiled rice, boiled macaroni, plums, bananas, almonds. 40 *per cent.* in : Chestnuts.

FOOD, 100 GRAMS.	PROTEIN.	FAT.	CARBO-HYDRATE.	APPROX. CALORIES.
Beef, mutton, fowl, fish uncooked . . .	20	5 -10	—	125–170
Ham	20	25	—	300
Bacon	12	50	—	500
1 egg, about 50 grams without shell . . .	6·5	5	—	75
Milk	3	24	5	70
Good cream	3	30	3	200
Very thick cream	3	40	3	400
Butter	1	85	—	800
Cheese . . . . . . •	25	33	12	400
Bread	9	1	60	275
Wheat flour	12	—	75	350
Rice	8	—	80	350
Oatmeal	16	7	66	375
Potato	2	—	20	90

Vegetables lose carbohydrates in cooking, especially if the water is changed thrice. It is approximately correct to consider a mixture of these in the 5 per cent. groups as containing 1 gram to the ounce.

       1 gram albumen   contains 4 calories
             „ carbohydrate   „   4 „
             „ fat   „   9 „
             „ alcohol   „   7 „
       1 kilogram = 2·2 pounds
       30 grams or c.c. = 1 ounce
A patient at rest requires 30 calories per kilogram of body weight per day.

VIII. **Predigested Foods** are indicated in dilatation of the stomach, cancer, and advanced cases of chronic gastritis. Benger's *Liquor Pancreaticus* is the usual ferment employed, because the pancreas contains both a proteolytic and a diastatic ferment. *Taka-diastase* is a valuable aid in the digestion of farinaceous foods. The patient takes it with his food at the commencement of the meal.

1. *Peptonised Milk.*—A pint of milk is diluted with a quarter of a pint of water and heated to a temperature of about 140° F. Two teaspoonfuls of Liq. Pancreaticus, with 20 grains of sod. bicarb., are mixed with it. The mixture is poured into a covered jug, and the jug is placed in a warm situation, in order to keep up the heat. At the end of an hour or an hour and a half the product is raised to the boiling point. It can then be used like ordinary milk. Peptonising powders are now to be obtained.

2. *Peptonised Beef-Tea.*—Half a pound of finely minced lean beef is mixed with a pint of water and 20 grains of sod. bicarb. This is simmered for an hour. When it has cooled down to a lukewarm temperature, a tablespoonful of the Liq. Pancreaticus is added. The mixture is then set aside for three hours, and occasionally stirred. At the end of this time the liquid portions are decanted and boiled for a few seconds.

(3) *Other foods* can be similarly prepared.

4. *Nutrient Enemata.*—Glucose alone is of practical use.

IX. **Tapioca Soup with Cream.**—Take a pint of white stock and pour into a stewpan. When it comes to the boil, stir in gradually 1 ounce of prepared tapioca. Let it simmer slowly by the side of the fire until the tapioca is quite clear. Put the yolk of two eggs into a basin, with two tablespoonfuls of cream. Stir with a wooden spoon, and pour through a strainer into another basin. When the stock is cooled, add it by degrees to the mixture, stirring well all the while, so that the eggs may not curdle. Pour it back into the stewpan, and warm before serving. Add pepper and salt to taste.

X. **Beef-Tea.**—Cut up a pound of lean beef into pieces the size of dice; put it into a covered jar with 2 pints of cold water and a pinch of salt. Let it warm gradually, and simmer for a couple of hours, care being taken that *it does not boil*.

XI **Improved Beef-Tea.**—Three-quarters of a pound of steak, scraped or passed through a mincing machine, and pounded; ½ pint of cold water; one piece of sugar, one pinch of salt, one teaspoonful of tapioca; simmered in a "Gourmet Boila" for three hours.

XII. **Artificial Proteid Foods.**—Beef-tea and other meat preparations do not contain the nutritive constituents of meat, except in small quantities, but may be useful as stimulants of gastric secretion. *Peptonised albumin* (or peptonised meat) is more nourishing, but the taste of peptone is very bitter and nasty. The *albumoses* are intermediate between albumin and peptone. They are freely soluble, tasteless, and readily digested and absorbed. *Plasmon* is another artificial protein food. It is prepared from milk, and contains casein in a soluble form. It is a nutriment of some value.

XIII. **Milk, Egg, and Brandy.**—Scald some new milk, but do not let it boil. Put it into a jug, and the jug into a dish of boiling water. When the surface looks filmy, it is sufficiently done, and should be put away in a cool place in the same vessel. When quite cold, beat up a fresh egg with a fork in a tumbler, with a lump of sugar; beat quite to a froth, add a dessertspoonful of brandy and fill up the tumbler with scalded milk.

XIV. **Chicken Panada.**—Take the flesh from the breast of a freshly roasted chicken; soak the crumb of a French roll or a few rusks in hot milk, and put this into a clean stewpan, with the meat from the chicken reduced to a smooth pulp by chopping it and pounding it in a mortar; add a little chicken broth or plain water, and stir the panada over the fire for a few minutes.

XV. **Whey.**—Into a vessel of warm milk put sufficient quantity of rennet to cause curdling, and strain off the liquid, which is then ready for use.

XVI. **White Wine Whey** (especially good for infants with summer diarrhoea).—Half a pint of milk is boiled: as soon as it boils, add 2¼ fluid ounces of good sherry; allow the mixture to boil for a few minutes, then leave in a cool place in a basin. When the curd falls to the bottom, carefully pour off the whey, or strain through muslin. In grave conditions, with vomiting, give a teaspoonful every ten minutes; in inflammatory diarrhoea give a tablespoonful every hour.

XVII. **Artificial Feeding of Infants.**—GENERAL DIRECTIONS.—Feed the child regularly; if necessary, wake it for that purpose. Use a boat-shaped bottle, with a rubber teat on the end. Feed slowly, holding the bottle on the slope until the milk in it is finished. Keep the bottle strictly clean by scalding it both before and after it is used. Mix a fresh portion for every meal. Do not overfeed; 2 pints of the mixture in twenty-four hours is enough for a child under six months. No starchy food should be given to an infant under six months, for the pancreatic secretion is not established till then. A baby after nine months requires other food as well as the breast, and should be weaned between the tenth and twelfth month. The best substitute for breast milk is the mixed milk from a good dairy herd, preferably certified Grade A. When the milk supply is not reliable condensed or dried milks may be used, such as Cow and Gate, Glaxo or Lactogen; the vitamins destroyed in the preparation may be replaced by giving orange juice.

UNDER ONE MONTH.—Feed every two hours from five in the morning to eleven at night. Start with ¼ ounce of milk to ¾ ounce of water, and gradually increase to 1¼ ounces. Sugar should be added in the proportion of 1 drachm of milk sugar to 4 ounces of the prepared milk. A small teaspoonful of cream may be given with each feed. When the warm mixture has cooled down, a teaspoonful of lime-water may be added.

FROM ONE TO THREE MONTHS.—Feed from 6 a.m. to 10 p.m. every three hours, with quantities gradually increasing up to 1½ ounces of milk to 2 ounces of water.

FROM THREE TO SIX MONTHS.—Feed every three hours with 3 ounces of milk to

2 ounces of water, gradually increasing strength to 4 ounces of milk with 3 ounces of water. The average amount is 1 ounce of milk for every month of life.

FROM SIX TO NINE MONTHS.—Feed every four hours. Milk, 6½ to 8 ounces, with water, 1 to 2 ounces. Later, add a little bread and milk, porridge, or pudding once or twice a day.

FROM NINE TO TWELVE MONTHS.—The bottle may be gradually left off. Morning and evening, 6 ounces of bread and milk, sweetened. Lunch.—Milk and water, bread and butter. Dinner.—Two ounces of farinaceous milk and egg pudding on alternate days; a little broth or beef-tea with bread on other days, or meat gravy.

FROM TWELVE TO EIGHTEEN MONTHS.—Morning and evening, about 6 ounces of bread and milk, sweetened, and bread and butter. Lunch.—Half a pint of milk, bread and butter. Dinner.—Bread, vegetables, milk pudding, and milk and water. On alternate days give gravy or broth, with bread-crumb, and milk pudding. Tea.— Bread and butter, and milk. Half an egg may be given once a day.

FROM EIGHTEEN MONTHS TO TWO YEARS.—In addition to the last-named diet, give minced meat or fish on alternate days, with finely chopped greens and potatoes. At teatime, cocoa. Mutton and bacon fat, finely chopped, and raw meat juice, are to be recommended for delicate children.

A useful addition to methods of infant feeding is the use of sodium citrate, 2 grains to the ounce of milk. In this way undiluted milk may be used, so that no cream or sugar need be added. Many doctors now feed infants from one month old with undiluted cow's milk. See remarks on Vitamins in §§ 440 and 478.

CHAPTER XI

THE INTESTINAL CANAL

THE physiological importance of the intestinal canal is evidenced by the fact that its length is between 25 and 30 feet, along the whole of which absorption may take place ; yet the first feature of intestinal disorders which strikes the student is their inaccessibility to examination. Bacilli or their toxins can make their way through the mucous membrane of the intestine into the lymph spaces beneath, and thence into the glands and the circulation, particularly when the mucous membrane is unhealthy, abraded, or ulcerated ; thus intestinal sepsis constitutes a danger for long but little realised. The bacteriology of the intestinal canal is now assuming much importance, and the examination of the stools is necessary in every complete investigation of a case.

Another striking feature about diseases of the intestines is the disproportionate amount of prostration which accompanies them. For instance, in a patient who is attacked by a slight but sudden diarrhœa or abdominal pain, the feeling of exhaustion, which in some cases may amount almost to collapse, is out of all proportion to the local mischief. This disproportionate degree of prostration or collapse is especially marked in early life, when " diarrhœa " is, mainly on this account, found to be the principal cause of death in children under two years of age. Again, among the acute specific fevers we find that the most fatal collapse and prostration occur in those in which the chief lesion is in the intestinal canal—in cholera, dysentery, and enteric fever. These facts are possibly accounted for by the circumstance that the chief centre of the sympathetic system (its " brain " so to speak) is found within the abdominal cavity, in close anatomical relation with the intestines which it supplies with nerves.

PART A. SYMPTOMATOLOGY

§ 237. The cardinal symptoms of intestinal disorder are DIARRHŒA, CONSTIPATION, and ABDOMINAL PAIN.

ABDOMINAL PAIN is frequently present, especially in the more acute conditions, but by no means always ; and abdominal pain may be due to so many other diseased conditions within the abdominal cavity that it has been considered in Chapter IX (the Abdomen, § 191).

DIARRHŒA is a cardinal symptom of intestinal disorders, and it will be fully discussed in Part C. of this chapter.

The same remarks apply to CONSTIPATION, and in this instance we shall have to distinguish simple constipation from that important surgical emergency, Obstruction of the Bowels. This also will be dealt with in Part C.

The GENERAL or REMOTE symptoms are sometimes, especially in acute cases, of a very severe character, in view of the profound PROSTRATION which is associated with some intestinal disorders—to which allusion has just been made. PYREXIA is not usually a marked feature in intestinal diseases (see § 189). In the more chronic forms of intestinal disease EMACIATION is apt to ensue from malnutrition. The sallow SKIN of intestinal toxæmia is well known. Various NERVOUS DERANGEMENTS of a neurasthenic order are sometimes, as in gastric diseases, associated with disorders of the intestinal canal, consequent partly on mal-assimilation and intestinal toxæmia, and partly, no doubt, arising in a reflex manner. Reflex symptoms of a less troublesome order—e.g., vague pains, itching of the nose, or bad dreams—may be associated with intestinal parasites and some other intestinal conditions.

PART B. PHYSICAL EXAMINATION

§ 238. The physical investigation of the intestinal canal can only be accomplished by two means, the EXAMINATION OF THE ABDOMEN and the INVESTIGATION OF THE FÆCES. A complete X-ray examination and examination with the sigmoidoscope is called for in some cases.

The **Examination of the Abdomen** is not always easy, but it should never be neglected in suspected intestinal disorders. PALPATION and PERCUSSION will enable us to make out any generalised swelling or localised tumour. The tenderness which often accompanies intestinal disorders may also be elicited. One can often feel a loaded caecum or the *scybala* present within the colon; these must not be mistaken for the nodules of cancer or other tumour. Their mobility is a very deceptive feature, and the occasional association of diarrhœa may delude us. Their disappearance after active purgation is the only certain method of diagnosis. The reader is referred to § 190 for further details as to examination of the abdomen.

§ 239. An **Examination of the Stools** is always important, and sometimes absolutely necessary for the diagnosis of intestinal disorders. A great deal of information can also be thus obtained with regard to diseases of the other abdominal viscera. The fæces should be examined *first* as to their physical properties—colour, consistence, shape, size, odour, and reaction; *secondly*, for undigested food and other substances, such as mucus, gall-stones, or parasites; *thirdly*, for the presence of blood; *fourthly*, a microscopic examination is often necessary. Lastly, culture of the stools is in many cases of great value. One can rarely rely implicitly

on a patient's statement, even as to the colour and appearance of the stools ; and, however disagreeable it may be, we should, when thoroughness is desired, examined the fæces ourselves. Much work has been done of recent years on the examination of the fæces—bacteriological, chemical, and microscopical—by Herter, Schmidt, Strasburger, and Cammidge. Early disease of the pancreas and of the intestinal canal can be detected by the thorough investigations which, however, can be carried out only in a properly equipped laboratory. For the technique of these examinations the student should consult the original writings and modern bacteriological text-books.

A small portion of the fæces may be brought in a tin box, but it is preferable to see them in bulk, the patient having used a night-stool. He should pass water before going to stool. A large wide-mouthed glass jar, closed at the top by a stopper, is a very convenient receptacle for their preservation. Nothing should be added to the motion until the doctor has examined it. Then carbolic solution (1 in 100) may be added if we wish to detect mucus or to preserve the stool.

Physical Properties of the Stools.—1. The COLOUR of the fæces is normally dark brown. The degree of colour of the stools is a fair measure of the amount of bile which passes into the intestinal canal. When there is diarrhœa the stools, at first, are dark from excess of bile pigments ; afterwards they become lighter on account of their dilution by the increased watery exudation and the presence of undigested food. (i.) *Clay-coloured stools* are found in cases of OBSTRUCTIVE jaundice, and pale bulky stools are also found with defective pancreatic secretion in advanced cases. (ii.) *Streaks of blood* may be present. (iii.) *Tarry stools*, of a dark or black colour, are due to the presence of blood which, entering the alimentary canal *high up* (as in cases of gastric ulcer), has undergone " digestion." (iv.) *Black fæces* are seen when the patient is taking iron, bismuth, or manganese internally. (v.) Colourless " *rice-water* " or *milky stools* are met with in cholera, severe dysentery, or severe entero-colitis, due chiefly to the presence of serum. (vi.) In infancy the stools are normally of an *orange-yellow* colour, but in " dyspeptic " diarrhœa or enteritis they are generally *green*. (vii.) Frothy stools, the result of bacterial decomposition, are seen characteristically in sprue.

2. THE CONSISTENCE OF THE STOOLS is normally semi-solid, and THE FORM is that of a rounded cylinder. (i.) When passed in hard, dry, roundish balls they are known as *scybala*. These are generally coated with mucus. Sometimes the irritation they cause sets up a false diarrhœa, and there are alternating conditions of diarrhœa and constipation, which can only be cured by a course of aperient medicines. (ii.) In typhoid fever the stools often present the appearance of pea soup. The *rice-water* diarrhœa of cholera has just been referred to. (iii.) In cases of stricture of the rectum—*e.g.*, from syphilis or cancer—the stools are *ribbon-like* in shape and this forms an important diagnostic indication.

3. The ODOUR of the stools is due to skatol and indol. There is a characteristic gangrenous odour in severe ulceration—syphilitic, cancerous.

or dysenteric. An ammoniacal odour is never met with in human fæces. If this odour be present it can only arise from the presence and decomposition of urine.

4. The REACTION of the stools on a mixed diet is normally feebly alkaline when first passed; with excess of meat, distinctly alkaline; with excess of starchy foods and fats, distinctly acid. Putrefaction and fermentation increase the alkalinity and acidity respectively after passing, so that the reaction ought to be taken as soon as possible. In pancreatic disease the reaction may be acid. The simplest test is to moisten red and blue litmus papers with distilled water, and rub a small portion of the stool on the paper. The reaction is best seen on the other side.

VARIOUS SUBSTANCES may be found—

1. UNDIGESTED PARTICLES OF FOOD, if in excess, are indicative of imperfect digestion (gastric or intestinal), and, unless the food has been
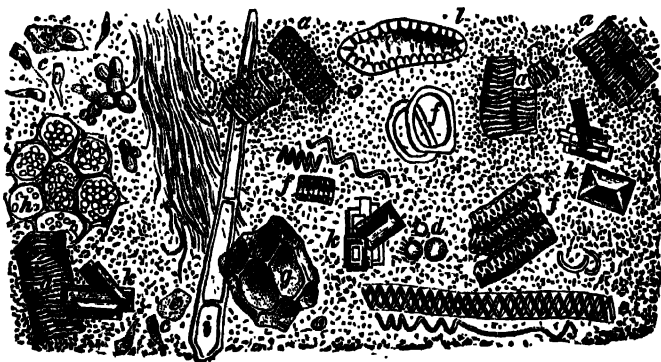
FIG. 60.—MICROSCOPICAL EXAMINATION OF FÆCES (after Von Jaksch).—Normal appearance × about 350. *a,* muscle fibres; *b,* connective tissue; *c,* epithelium; *d,* white blood cells; *e,* spiral vegetable cells; *f* to *i,* various vegetable cells; *k,* triple phosphate crystals in a mass of micro-organisms; *l,* diatoms.

excessive, denote especially intestinal or pancreatic disease (see also p. 280). In children this feature usually indicates over-feeding. Small, hard concretions, consisting of phosphates and other matter, are sometimes found. By noting carefully which articles of diet (proteid, vegetable, fruit, or carbohydrate) pass for the most part undigested, the physician learns which the patient should be forbidden to eat.

2. MUCUS in the fæces is often overlooked unless specially sought for. To discover it satisfactorily *water must be added* to the fæces, when any mucus present will be seen floating about like small pieces of jelly. The presence of mucus in small amount is of no consequence ; it is usual in constipation. When in quantity, and intimately *mixed with the fæces,* it indicates catarrh of the *small* intestine. When in *isolated masses* it signifies the presence of catarrh of the large bowel. In membranous, or mucous, colitis, *long cylinders* of mucus are passed, sometimes without much fæces.

C.M.

These cylinders are generally swarming with organisms of both coliform and streptococcal types, which infest the colon.

3. BLOOD in the stools may appear either in streaks or in quantity, when from rectum or large bowel. If it comes from the stomach or small intestines, it will have undergone partial digestion and give to the stools a tarry appearance (melæna). In either case it reddens the water in which the stool is placed, and gives the characteristic spectrum.[1] The *causes* are dealt with below (§ 249). *Occult* blood must be tested for in cases of suspected oozing from an ulcerated surface. A simple test for occult blood is the Benzidin test which is stated to give a positive reaction with one part blood in 250,000. A small amount of stool is emulsified and boiled. Meantime a knife point of benzidin is dissolved in 1–2 c.c. acetic acid (glacial). Five to ten drops of the boiled and cooled emulsion are diluted with an equal volume of water, and this is added to the benzidin solution, and the whole is well mixed. Hydrogen peroxide is added drop by drop until a blue colour appears, but not more than 2 c.c. is used. Depending on the amount of blood present the blue colour appears at once or after a few minutes. For three days before the test, no meat or green vegetables must be eaten. A series of tests over consecutive days is of importance where the result is positive. If each is positive the diagnosis is in favour of a malignant ulceration ; if the positive result is intermittent it is in favour of simple ulceration.

4. PUS always indicates *ulceration* of the rectum or colon, which may be of syphilitic, cancerous, tuberculous, or dysenteric origin (§ 246). Pus is difficult to detect when diarrhœa is present. When in large quantity, pus indicates an abscess bursting into the bowel, such as a pelvic or ischio-rectal abscess.

5. GALL-STONES may be found by mixing the stools with water, and passing the mixture through muslin or a fine sieve. Gall-stones sink in water when recently passed, though they float when dried. They are very friable, and any suspicious particles should be examined under the microscope for cholesterin, see Fig. 69.

6. WORMS, such as tapeworms and threadworms, may be found. It is of great importance to find the head of the tapeworm. It is about the size of a pin's head. This may be done by the method just described. Another method is, to mix the fæces with water and let the mixture stand. As the parasite sinks to the bottom the supernatant fluid should be carefully poured off, and more added, the process being repeated until the fluid becomes colourless. The various worms are given in the Table XVII. The larvæ of flies are occasionally found.

Microscopic Examination of the fæces is often necessary, especially to find the ova of parasites. Place a small portion of the stool upon a slide, and if not sufficiently

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[1] Cases have been recorded where, after standing for some time, the fæces developed on the exposed surface a colour resembling blood, but no blood was detected by the spectroscope. It appears that in certain as yet unknown conditions some pigment is present in the fæces, which on exposure to the air becomes red like blood.

fluid, dilute with a quantity of normal saline solution ; mount a second portion in iodine ; a third smear is dried, fixed and stained by Gram's method.   If dysentery is suspected, the stool must be examined whilst still warm.   NORMALLY, under the

FIG. 61.—HEAD OF TÆNIA MEDIO-CANELLATA. Magnified about 10 times.

FIG. 62.—TÆNIA SOLIUM. Head × 30 (a) ; and proglottides (b) or segments (slightly enlarged).   In the latter the uterus has seven to ten lateral branches which ramify.   But in T. Mediocanellata there are twenty to thirty lateral branches, which in turn terminate in two branches (dichotomous branching).

FIG. 63.—BOTHRIOCEPHALUS LATUS. Natural size.   Head (a) ; proglottides (b) ; and tail (c).

FIG. 64. — Female OXYURIS VERMI-CULARIS. a, natural size, about ⅓ inch.
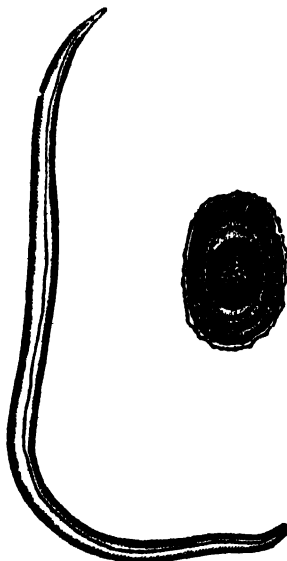
FIG. 65.—ASCARIS LUMBRICOIDES (Round Worm). About half normal size, and EGG × 75.

microscope (Fig. 60), the stool shows undigested particles of food, especially starch, granules, muscle fibres, connective tissue, and fat cells ; crystals of fatty acids, oxalate of lime, and other calcium salts.   Hæmatoidin, phosphates, cholesterin, and Charcot-Leyden crystals are rare.   Among the bacteria the Bacillus coli communis, various

unnamed bacilli, cocci, and yeast are found. Blood corpuscles and intestinal epithelial cells may occur in small amount.

Among the ABNORMAL constituents which should be looked for are, *first* and chiefly the presence of the ova or segments of the different entozoa (see below, § 240).

2. Among the *undigested food products* an excess of undigested starch or of muscle fibre indicates disease of the small intestine or pancreas. The starch is stained a deep blue in the Iodine slide : Where the starch is excessive iodine staining bacilli are often numerous   An excess of fat in the faeces indicates (i.) deficient bile secretion, (ii) disease of the pancreas, or (iii.) intestinal disease interfering with fat absorption. The relation of unsaponified to saponified fat is normally about 10 to 15 per cent. In pancreatic disease the unsaponified fat is in excess.   In biliary obstruction the saponified fat is in excess.   In cancer of the pancreas, with jaundice, the relations are about equal.   *Intestinal sand* consists of fine granules like sand, due to calcium

FIG 66 —TRICHURIS TRICHIURA (Trico-cephalus Dispai.   "Whip-worm") — Magnified by 3, and EGG magnified about 100.

OVUM DUODENALE (and female) —Woim egg × 175. *a*, natural size The lower one is from a micro-scopic specimen for which the author is indebted to Dr. W. J. Tyson, of Folkestone.

salts and silica forming around an organic nucleus, or to granules from pears and other fruits.

3. The *Charcot-Leyden* crystals are the only abnormal crystals of any importance. They are very rare, and are found chiefly in association with worms, especially anky-lostomum and mucous colitis.   Their presence is a useful indication that the parasite is still alive in the intestinal canal.   Excess of fatty acid crystals is found with pan-creatic disease.

4. Various *bacilli*, such as those of the typhoid group, dysentery and cholera, are present in the faeces in disease, but their isolation requires cultural methods for their recognition.   The *b. coli* has its normal habitat in the colon.   Streptococci of various types are present and in some cases in enormous numbers.   Staphylococci, aureus and albus, are occasionally found : and S. citreus is present in Still's Disease.   Abrupt alterations in diet are followed by rapid alterations in the prevailing types of bacteria, and this is true also after the administration of lactic acid bacilli in certain putrefactive conditions.

§ 240. Various Intestinal and Other Parasites, or segments of them, or their ova,

may be found in the fæces. These are described in Table XVII, and the accompanying illustrations. Seven of these *infest the alimentary canal of man :* The two common tapeworms (T. Solium and T. Mediocanellata), recognised by their segments in the fæces, naked eye; the tapeworm of Central Europe (Bothriocephalus Latus), recognised by its segments, naked eye; the extremely common threadworm (Oxyuris Vermicularis); and the common round worm (Ascaris Lumbricoides), both of which may be seen by the naked eye, the former like small pieces of cotton, the latter as large as a garden worm; and two worms which are chiefly found abroad, the Ankylostomum and the Tricocephalus, both of which, with their ova, need magnification for discovery. The ova of various forms of Distoma may also be found in the fæces (§ 441). The symptoms and treatment of the common worms are given in § 251. The eggs of the S. mansoni are sometimes found in the fæces, enclosed in small fleshy masses; their ova have a lateral spike. The ova of the S. hæmatobium (Bilharzia) are found in the urine, associated with *hæmaturia* (§ 300). The ova have a spine-like projection at one end (Fig. 87).

The *amœba* of amœbic dysentery was first found in the stools in 1875. It is difficult to distinguish from the harmless scavenger, the *entamœba coli.* The *entamœba histolytica,* the true dysenteric amœba, can only be recognised if it contains red blood cells. The cysts of both parasites are characteristic; those of the *entamœba coli* showing eight nuclei, those of dysentery only four. The amœbæ are generally found in a drop of freshly voided mucus in fair abundance as roundish cell-like bodies of irregular oval form, which continually undergo amœboid movements on a warm slide.

Cysts of *Lamblia Intestinalis* are more frequently seen than one would expect. They do not seem to have any special significance except that, since their habitat is high in the small bowel, their presence indicates a possible derangement of the small intestine.

PART C. DISEASES OF THE INTESTINAL CANAL, THEIR DIAGNOSIS, PROGNOSIS, AND TREATMENT

241. Routine Procedure, and Classification.—Having first ascertained that the patient's LEADING SYMPTOM is referable to the intestinal canal; and secondly, by inquiries into the HISTORY of the illness, whether it came on *acutely* and suddenly or gradually in a *chronic* manner; we proceed, in the third place, to the PHYSICAL EXAMINATION of the abdomen after the manner set forth in Chapter IX (§ 190). If, in the course of these inquiries, definite disease is suspected in any particular organ, reference should afterwards be made to the appropriate chapter.

A. If **Diarrhœa** is the leading symptom:

If *acute*, or attended by choleraic or dysenteric symptoms   ..   ..   turn to §§ 243-245

If *chronic*   ..   ..   ..   „   § 246

B. If there is **Tenesmus** without diarrhœa   ..   „   § 248

C. If **Blood** or some other **alteration** in the stools is the leading feature   ·   ..   ..   „ §§ 249-251

D. If **Constipation** is the leading symptom   ..   „   § 252

E. If the **Stoppage in the Bowels** is complete   ..   „   § 253

§ 242. Diarrhœa is the frequent occurrence of loose or liquid motions; it is the watery consistence of the stools which is the chief characteristic in diarrhœa. A frequent call to stool may arise from some local irritation (see Tenesmus), without any alteration in the consistence or form of the

TABLE XVII. THE PRINCIPAL ENTOZOA.

NAME.	CHIEF CHARACTERISTICS OF ANIMAL, AND WHERE FOUND.	CHIEF SYMPTOMS.	OVA OR EMBRYO; CHIEF CHARACTERISTICS, AND WHERE FOUND.	ANIMAL HOSTS, ETC.
FAM.: CESTODES. Tænia Medicanellata. (Tapeworm in man.) Fig. 61.	14 to 24 ft. long. Head, 4 suckers, no hooklets. Segments, over 1000, show central stem uterus with 20 to 30 lateral *dichotomous* branches. Fastens itself to mucous membrane of intestinal canal in man.	Of reflex irritation, digestive or nervous disorders. Segments passed per rectum.	Recognised by segments containing ova discharged from alimentary canal in faeces. Ova more oval than in T. solium. Embryo found in beef.	Cattle the intermediate hosts. Found in Great Britain.
Tænia Solium, Fig. 62. (Tapeworm in man.)	At about 10 ft. long. Head, 4 suckers, and row of 26 hooklets. Segments, about 850, show central stem uterus with 7 to 10 lateral *ramifying* branches. Fastens to mucous membrane of intestinal wall in man.	Ditto.	Recognised by segments containing ova discharged per rectum. A six-hooked embryo inside ovum; which eaten by pig, bores its way into the flesh.	Pig the intermediate host—mostly pork," where scolices abound in flesh. Animal takes 3 months to develop in man.
Bothriocephalus Latus. Fig. 63.	16 to 25 ft. long. Head club-shaped, with long lateral slits. No hooklets or suckers. About 3000 segments; uterus, rosette-shaped. Found in intestinal canal of man.	Very few symptoms in adults. Intestinal disorder in children.	Segments containing ova, discharged per rectum. Sometimes ova discharged alone; brown shelled; ⅛ in. long; with a lid at one end.	Ova hatched on reaching water, and eaten by fish, which act as intermediary host. Chiefly found in Switzerland and other parts of Central Europe.
Tænia Echinococcus. (Hydatid cyst in man; tapeworm of dog.) Fig. 74.	⅛ in. to ⅓ in. long. Head pointed, with 4 suckers; double row of hooklets. Has 4 segments, the 4th longer than all others. Found in intestinal canal of dog or wolf.	Hydatid cysts form in liver, or other organs, in man.	Ova found in faeces of dog or wolf. Embryo reaches man by drinking water, and becomes encysted in various organs.	Man is the intermediate host; dog or wolf—the host. Man receives the embryo by drinking contaminated water—*e.g.*, mountain streams.
FAM.: NEMATODES. Oxyuris Vermicularis. (Threadworm.) Fig. 64.	F. = ⅓ in.; M. = ¼ in. in length. Found in large intestine, chiefly the rectum.	Reflex irritation. Worms tend to migrate at night, and cause itching of anus and genitals.	Worms easily seen after aperients.	Often trouble children. Found in all countries.

Ascaris Lumbricoides. (Round worm.) Fig. 65.	M. about 6 in. long; F. 12 in. Found in small intestine of man.	Reflex irritation, nervous and digestive.	Ova, $\frac{1}{50}$ in. to $\frac{1}{70}$ in. in diameter. Hard, dark shell with excrement. Found in faeces.	Worms tend to migrate, and are sometimes vomited. Frequent in children. Are found in all countries.
OTHER FAMILIES. Trichuris trichiura. Fig. 66. (Trichocephalus Dispar.)	1½ in. long. Anterior part fine and thread-like. Found in caecum.	Few symptoms.	Ova elliptical, with a projection at each end. Dark coloured, $\frac{1}{50}$ in. long. Found in faeces.	Cosmopolitan.
Ankylostomum Duodenale. Fig. 67.	F. = $\frac{1}{2}$ in.; M. = $\frac{1}{3}$ in. Tail end broadest; mouth capsule distinct and provided with 4 tentacles, which clasp the villi of the small intestine. Found in jejunum.	Melena, profound anaemia and weakness; urticaria and boils. Eosinophilia.	Ova about $\frac{1}{50}$ in. Clear and transparent shell, showing yolks through. Worms found in faeces.	Found in India, Egypt, Brazil, Jamaica, and Mining districts.
Filaria Sanguinis Hominis. (Filaria Bancrofti.) Fig. 118.	3 or 4 in. long; fine and hair-like, lying in bunches together. Found in lymphatics.	Chyluria; Elephantiasis.	Embryos $\frac{1}{8}$ in. long, enclosed in thin envelope. Tapering tail and round head. Found in blood at night.	Mosquito acts as intermediate host, by taking embryo from blood. Chiefly exist in India and tropics.
Schistosoma haematobium. (Bilharzia). S. mansoni; S. japonicum. Fig. 87.	M. = 15 mm. long; cylindrical, with gynaecophoric canal. F. = 20 mm. Found in blood of portal system, &c.	Haematuria and melaena; debility and anaemia.	Ovum has one terminal or lateral spine. Oval, 0·16 mm. long. Found in bladder and urine, and in faeces.	Intermediate host a fresh-water mollusc. Man gets parasite by water. Egypt, Africa, W. Indies, the Far East, and Brazil chief countries.
Trichina Spiralis.	M. = $\frac{1}{12}$ in.; F. = $\frac{1}{8}$ in. Lives a few weeks in intestine of man, and then embryos migrate to muscles.	1st stage, gastro-intestinal irritation; 2nd stage, fever, tenderness of muscles, oedema. Often death in 5 weeks.	Larval form bores through alimentary canal, and is found coiled up within a cyst, in muscular tissue. Adult worm and occasionally embryo found in faeces. Eosinophilia.	Man gets parasite from uncooked pig flesh. Rabbits, mice, rats, sheep also get the parasite. Found in Germany; rare in England.
Filaria Medinensis. (Guinea worm.)	F. = 13 in. to 30 in. Cylindrical, white, smooth. Found in subcutaneous tissues of limbs, especially the feet.	Local inflammatory symptoms; later constitutional symptoms.	Embryo flattened, $\frac{1}{30}$ in. long, with long thin tail.	Presumably acquired by drinking water, which contains intermediate host. Found in Asia, Africa, Egypt, Brazil. Intermediate host—a fresh-water cyclops.

stool. This source of fallacy should be carefully guarded against. Many women speak of the tenesmus which sometimes accompanies the menses as "diarrhœa."

CAUSES OF DIARRHŒA.

	Acute.		Chronic.
COMMON.	I. Irritating food.		I. Acute causes becoming chronic.
	II. Water.		II. Local conditions about anus.
	III. Intestinal parasites.		III. Ulceration (colitis, tuberculosis,
	IV. Infantile diarrhœa.	COMMON.	cancer and syphilis of the
	V. Typhoid and other toxic blood		bowel).
	conditions.		IV. Chronic colitis or mucous colitis.
	VI. Acute enteritis or "chill."		V. Portal obstruction or congestion.
RARE.	VII. Acute ulcerative colitis.		VI. Dysenteric diarrhœa.
	VIII. Some causes of chronic		VII. Nervous diarrhœa.
	diarrhœa.		VIII. Amyloid disease.
	IX. Dysentery.	RARE.	IX. Senile diarrhœa.
	X. Cholera.		X. Mineral poisons (e.g., arsenic).
			XI. Pancreatic disease.
			XII. Psilosis (Sprue).

The fæces should always be examined where it is possible (§ 239). Sometimes the situation of the disease may thus be discovered; for instance, when the stools are coloured with bile, and contain undigested food, and *small pieces of mucus intimately mixed* with the fæces, catarrh of the small intestine may be suspected. When mucus or "slime" occurs in *larger masses*, in "strings" or "casts," there is probably disease of the large intestine.

§ 243. In **Acute Diarrhœa** there is usually a good deal of pain and tenesmus (straining at stool); the tongue is usually furred, there is thirst, and may be vomiting. If there be much vomiting and prostration, the diarrhœa is probably due either to the presence of some violent irritant, or to some serious organic lesion, such as injury to the bowel or peritoneum. In profuse diarrhœa the temperature is usually subnormal, and the urine diminished. It should be borne in mind that scybala retained in the intestines may give rise to attacks of diarrhœa alternating with constipation. The possibility of a controlling appendix is also to be considered.

Causes.—I. The **food** taken, and the vessels in which it has been contained and cooked, should be the first questions in all cases of acute diarrhœa coming on suddenly in a healthy person. Collapse and many of the symptoms of cholera can be produced by food cooked in a new copper vessel. One of the irritant poisons may have been introduced into the food accidentally or designedly. This should be borne in mind; and in cases of sudden and unexplained diarrhœa the physician should patiently consider every article taken at every meal during the preceding twenty-four hours. Over-ripe or decomposing fruit, too much raw vegetable food, tinned meat—especially that which has been long in store and has undergone a change resulting in the formation of ptomaines—shellfish and bad cheese are also possible causes. In this variety of acute diarrhœa there may be a considerable degree of intestinal colic (§ 196).

The first or diarrhœal stage of trichinosis comes under this heading, and should be considered in pork-eating countries. In cases of acute diarrhœa in which trichinosis is suspected the worm should be sought in the fæces, for in the earlier stages of this disease treatment is so much more efficacious. The diarrhœa which precedes the intestinal obstruction caused by intussusception in children frequently follows a heavy meal of indigestible articles ; and diarrhœa is itself a cause of intussusception.

II. The quality of the water is often responsible for diarrhœa, acute or chronic. This is frequently the case in malarial districts in the summer and autumn, especially when the temperature is high. Water containing much peat from the mountains may also cause diarrhœa ; and thus the water supply of the town of Montreal frequently occasions diarrhœa in new-comers.

III Worms may give rise to diarrhœa in children. They may be attended by uneasy abdominal sensations, night terrors, picking of the nose, itching of the anus, but sometimes the worms are discovered in the stools when there have been no symptoms (§ 251). The presence of the *Lamblia Intestinalis* may be a cause of acute diarrhœa from time to time.

IV. Infantile Diarrhœa occurs in at least three well-recognised clinical forms : (i.) Acute Catarrhal or Dyspeptic Diarrhœa ; (ii.) Inflammatory Diarrhœa or Entero-colitis ; and (iii) Epidemic Diarrhœa or "summer diarrhœa" (including Infantile Cholera)– mentioned in progressive order of severity.

(i.) In ACUTE CATARRHAL (dyspeptic) DIARRHŒA the stools are offensive, at first yellow, then greenish, slimy and mixed with curds of undigested food. Vomiting may or may not be present. It is usually a transient condition if adequately treated.

(ii.) In INFANTILE INFLAMMATORY DIARRHŒA (Entero-colitis) the stools are green, slimy and often contain blood ; there is some fever at the beginning, and abdominal distension. The stools vary with the predominant infection ; they are acid and frothy in the fermentative type, alkaline and green in the putrefactive variety. The inflammation attacks chiefly the colon ; consequently there is tenderness on pressure over the region of the colon. In the stools mucus and blood occur in severe cases Prostration is great when much vomiting occurs. Adults also are sometimes affected. It lasts only one to three weeks if treated correctly

. (iii) EPIDEMIC DIARRHŒA ("summer" or "autumnal" diarrhœa of children) is met with chiefly in childhood and infancy in the autumn months of the year, and is attended by catarrh of the mucous membrane of the bowel. The *symptoms* of a severe attack are : Watery stools, foul-smelling, of altered colour, containing lumps of mucus , vomiting ; acute abdominal pain and tenesmus ; prostration, collapse, subnormal temperature with pinched aspect, rapid wasting, and often (after a course of a week or so) death from exhaustion. INFANTILE CHOLERA forms about 2 per cent. of "summer diarrhœa" cases. The stools are serous,

persistent vomiting is a marked feature ; great collapse rapidly super-
venes, the temperature in the rectum is raised as in adult cholera, and
death soon follows.

Etiology of Infantile Diarrhœa.—Any of the previously mentioned
causes (I to III) are contributory, and very often exciting, causes in all
forms of infantile diarrhœa ; and especially dietetic errors. This latter
is the sole cause in variety i , and probably in variety ii. These diseases
affect chiefly hand-fed and over-fed children, in warm weather, being,
probably in part due to dirty feeding-bottles, teats, sour milk, etc. Most
of the cases occur in children under six months old. The causes of *Epidemic
Diarrhœa* are far more obscure   Seasonal, epidemic, and microbic causes
have long been suspected on account of its prevalence during the summer
and autumn months. It occurs chiefly after hot, dry summers   Flies
and dust have been blamed, but no direct association can yet be traced.
It occurs chiefly in towns, and certain localities have been notorious for
recurrent lethal outbreaks in summer and autumn.   Ballard found that the
severity of the annual outbreak seemed to vary with the subsoil tempera-
ture ; it started when the 4 feet earth thermometer read 56° F.   Adults
do not altogether escape ; diarrhœa is widely prevalent in the hot, dry
summer months in some years , but in children the death-rate is sometimes
appalling

A rare form of severe Recurrent diarrhœa may occur in children, resembling the
Cœliac disease of older writers. The motions are pale and fatty, the abdomen is
distended. Owing to the continued defective absorption of food, the child does not
grow (infantilism) , yet after years of protracted illness fatal cases show no
organic cause to be in operation   Defective fat digestion appears to be the only
error. The slightest error in diet will bring about a relapse of severe diarrhœa which
may last many months   The diagnosis from tuberculous peritonitis may be difficult

In the *Treatment of Infantile Diarrhœa* astringents are not only useless,
but harmful.   Equal parts of lime-water and castor oil (F. 64), every two
or three hours, until the stools become healthy, is a most valuable pre-
scription   This must be combined with appropriate diet—avoidance of
carbohydrates in the fermentative type, and of proteins in the putre-
factive type.   In the latter, lactose and lactic acid bacilli are indicated ;
in the fermentative variety, sterilised and skimmed milk, whey and protein
milk.   The vomiting may be checked by giving only barley-water for
a time, and the usual stomach sedatives.   In mild cases castor oil followed
by bismuth, rhubarb, soda, and cinnamon, or small doses of grey powder
or thymol will effect a cure.   In *epidemic diarrhœa* and in severe inflam-
matory diarrhœa milk and fats must be avoided.   Rice, albumen and
barley water can be tried cautiously, in small feeds ; in other cases weak
veal and chicken broth or tea.   Where collapse is present large rectal or
subcutaneous injections of hypotonic saline are indicated.   Glucose can
be added.   Brandy, 20 drops, and ⅓ drop of liquor strychninæ hypodermic-
ally are the best stimulants, and the infant should be put into a warm

mustard bath or hot pack until the harsh dry skin becomes soft and elastic. If vomiting persists, it may often be checked or diminished by washing out the stomach. This is easily done by using a soft rubber tube with a large eye, and a funnel.

V. **Toxic Blood States.**—Typhoid fever is nearly always attended by diarrhœa ; it sometimes complicates measles, and the other eruptive fevers (especially at their advent). Graves' disease, chronic renal disease, uræmia, and pyæmia ; and sometimes it appears at the termination of acute conditions, as in pneumonia. It may also be one of the effects of dissecting-room poisoning. Gouty people are often subject to attacks of diarrhœa, which are of a conservative nature.

VI. A chill to the surface in some individuals will determine an attack of acute diarrhœa.

VII. **Acute Ulcerative Colitis** is usually of sudden onset, with diarrhœa, and abdominal pain occurring in paroxysms. The motions are dark, offensive, and contain mucus and blood. There is tenderness over the colon, especially over its ascending portion, which is usually distended. The tongue is furred at first, and the breath very offensive. Pyrexia may be present, about 101° to 102°. The commonest complications are perforation, peritonitis, profuse hæmorrhage, and anæmia. Death may occur from exhaustion in one or two months. The *ulcerative colitis of asylums* is usually more severe, with vomiting and rigor, and may terminate fatally in a few weeks. Both diseases affect females about middle life. It may in some cases be difficult at first to diagnose from Enteric fever.

VIII. In cases of acute diarrhœa in which the cause is obscure, reference should be made to the other **Causes** of **Chronic Diarrhœa**, any of which may from time to time give rise to an acute attack. **Dysentery** (§ 244), and **Cholera** (§ 245) are the commonest causes of diarrhœa in tropical climates, and are occasionally met with in this country.

Prognosis of Acute Diarrhœa.—The causes of acute diarrhœa are for the most part removable ; and though weakened by the attack, the patient generally makes a good recovery. Acute Epidemic Diarrhœa in children, however, is a most fatal affection, and it leads to a higher death-rate in infancy, in Great Britain, than any other disease, accounting for nearly 2,000 *deaths of infants under 1 year old annually in London alone.* The prognosis in any given case depends upon (i.) the cause ; (ii) the severity of the symptoms and the evidences of weakness (iii) the state of the hygienic surroundings ; and (iv.) the treatment. Infantile cholera is rarely recovered from. Dyspeptic diarrhœa may be cured in a few weeks, but if untreated, is apt to go on to subacute enteritis. Without treatment all forms of epidemic diarrhœa, even in adults, are serious. Should symptoms of prostration or collapse ensue, the outlook is bad ; but it is only at the two extremes of life that this disease is so grave. Ulcerative colitis is very serious ; if death does not occur from complications, it usually occurs from the exhaustion, anæmia, or relapses.

Treatment of Acute Diarrhœa.—The indications are (a) to remove any

irritating matters present in the intestinal canal ; (b) to ensure *rest* to the irritated parts ; and (c) to *check excessive exudation.* (a) Thus, a simple acute diarrhœa in an *adult* following the ingestion of bad food is cured readily by a dose of castor oil, ½ oz. (16), with tr. opii, ℥x. (0·6) ; or calomel. (b) Milk and bland food only can be taken ; soups and beef-tea are not advisable. In severe cases withstanding treatment, the diet may be restricted to raw meat juice. Simple cases of dyspeptic diarrhœa in children are readily cured by grey powder every night, and alkaline carbonates during the day. If the stools are slimy, bismuth is needed. (c) After the expulsion of all irritant matters, a mild astringent, such as chalk or Dover's powder, is beneficial, and bismuth to soothe the congested mucous membrane. Kaolin is useful because of its absorbent properties. Astringents are contra-indicated in the *early stages* of diarrhœa, especially when due to (i.) irritants ; (ii.) inflammation ; or (iii.) portal obstruction. Only when the diarrhœa threatens to become chronic do we require to use astringents, such as catechu, kino, pulv. cretæ aromaticus, mineral acids, hæmatoxylin, and tannin. Opium allays irritation and checks peristalsis ; it may be given as tr. opii or tr. chloroformi et morphinæ. Coto is a useful drug ; it acts by diminishing the intestinal secretion. If the stools are very offensive, calomel, charcoal, carbolic acid, and creosote are useful ; and a course of intestinal antiseptics may be given—salol gr. x. (0·6), benzo-naphthol gr. v. (0·03), lactic acid, dimol, kerol, etc. Lastly, when other means fail, rectal injections must be resorted to—opium with starch, or silver nitrate ; but these are useful chiefly when the disease is in the larger bowel. In all severe cases, absolute rest must be insisted upon, with warmth to the abdomen. In ulcerative colitis Dr. Wedgwood's mixture often does good : liq. hydrarg. perchlor. Ʒss., tr. ferri perchlor, ℥ xx., every four hours.

The patient, who is or has been living abroad, complains of severe DIARRHŒA, WITH BLOOD, MUCUS, *and perhaps* PUS *in the stools.* The disease is probably DYSENTERY.

§ 244. VIII. Dysentery is a form of diarrhœa attended by severe tenesmus and frequent stools, and generally with pyrexia, *due to ulceration of the large bowel,* and depends on the presence of one or more specific organisms. It is met with clinically in two forms—(a) *acute* and (b) *chronic.* Both are characterised by (i.) diarrhœa, (ii.) the passage of blood, and (iii.) of mucus, from the bowel.

(a) ACUTE DYSENTERY may be of sudden onset. The patient wakens in the early morning with a griping pain, and tenesmus, and during the day there may be from ten to sixty scanty discharges from the bowel, containing blood, mucus, epithelial cells, and later on they acquire the appearance known as the " toad-spawn " discharge. In other cases there is abdominal pain and malaise for a few days before the onset of the diarrhœa. There is more fever and toxæmia in the bacillary than in the amœbic form. In a favourable case the discharge ceases after a week or ten days.

(b) CHRONIC DYSENTERY may result from an acute attack, or it may be chronic from the onset. In the latter form the patient has a gradually increasing diarrhœa, the stools becoming frequent and scanty, with some tenesmus, and the passage of a little blood or flakes of mucus, the symptoms gradually becoming worse. Dysentery affects the rectum, sigmoid flexure, and descending colon. Sometimes the disease extends as far up as the cæcum, and may consist merely of a catarrhal state of the mucous membrane. Severer cases lead to *ulcerative colitis,* which may result in

thickening and cicatricial tissue, and stricture. The most severe variety is the sloughing or *gangrenous* form, when large sloughs come away with an offensive odour, and are liable to set up septicæmia, or to cause perforation of the bowel, or a fatal hæmorrhage. X-ray examination is of use in detecting ulcer formation.

Etiology.—Dysentery most often affects men, especially if intemperate. Amœbic Dysentery occurs in the tropics, where it is endemic, and is due to the presence of the Entamœba histolytica (§ 240). It enters the alimentary canal by the drinking water and by food contaminated by flies. It is more apt to affect unhealthy persons, and is predisposed to by any disease or abrasion of the alimentary canal, such as occurs after eating unripe fruit, a chill, the abuse of purgatives, and especially constipation. In heart and kidney disease, secondary diphtheritic enteritis may occur, which is known as dysentery, but the description above refers mainly to true dysentery. Shiga's or Flexner's bacillus is the cause of such cases as are not due to the amœba. The acute dysentery affecting our troops in the European war was of both bacillary and amœbic origin, complicated by secondary bacterial infection.

Diagnosis.—The diagnostic features are the presence of the Entamœba histolytica, the culture of dysentery bacilli from freshly-passed stools, and the agglutinative reaction to dysentery bacilli. In the amœbic form of dysentery there is usually a history of an insidious beginning, and there is a great tendency to the formation of liver and other abscesses. Acute dysentery may be mistaken for *acute diarrhœa,* from which it is differentiated by examination of the stools. *Acute enteritis* due to *malaria* may be regarded as dysentery, and unless the case be treated with quinine the patient will die ; the blood in such cases should be examined for malaria parasites. A diagnosis of chronic dysentery should never be made before local examination has excluded rectal cancer, polypus, piles, bilharzia, and, indeed, any of the other causes of diarrhœa (§ 242). Diarrhœa due to *ulceration,* occurring in a tropical climate, may be mistaken for chronic dysentery. The ova of Schistosoma hæmatobium (Bilharzia) may be found in small masses resembling polypi which, on being broken up and examined by the microscope, show the ova (§ 326). The patient may also have a history of hæmaturia.

Prognosis.—An attack of acute dysentery in a healthy person may pass off in a week or so ; but it requires care in a tropical country to prevent it passing on to chronic dysentery, a condition which is very difficult, often impossible, to cure. The bacillary form is extremely dangerous, and has a higher mortality than any disease except Kala-azar. With chronic dysentery acute exacerbations frequently occur, and the patient becomes anæmic and greatly debilitated by the constant loss of blood. Complications arising in the course of chronic dysentery are ulcers, with consequent periproctitis, amœbic abscess of the liver, cicatrisation with rectal stricture, peritonitis, multiple pyæmic abscesses, and pneumonia. In some epidemics of bacillary dysentery polyarticular arthritis is common about the third week of the attack.

Treatment.—The main indication in both acute and chronic dysentery is to give rest to the inflamed part. This in the *acute* form is accomplished by keeping the patient absolutely at rest in bed, with no food except white of egg, barley water, chicken broth, yaghourt, etc., for a few days. Emetine is the best remedy for amœbic dysentery. Emetine hydrochloride—½-¾ grain (0·13 gm.) is injected once or twice daily, and continued for a week or ten days. Emetine periodide is given at the same time by mouth—4 grains daily for 12 days. For bacillary dysentery a saturated solution of sodium sulphate (half an ounce, followed every 2 to 4 hours by 1 to 2 drachms (4 to 8 gm.)) is given until there is no more blood and mucus and no fever. It rarely requires to be given longer than 3 to 6 days. Hot fomentations are used for the abdominal pain. Opium and adrenalin in starch enemata relieve the tenesmus ; or the bowel may be washed out with a solution of calcium permanganate (gr. 6 to 1 pint (0·3-600)), organic silver salts or eyllin. Polyvalent serum should be injected early. Large initial doses, 80 to 120 c.c., are now given intravenously as early in the disease as possible for severe cases. Intravenous saline (sod. chlor., sod. carb. āā ʒj,

boiled water 1 quart) is called for in cases where much serous exudation is evacuated. In chronic dysentery the diet must be non-irritating, but it is not good to keep the patient too long on milk food. Sometimes the patient may recover rapidly on being sent a sea voyage. Constipation must be avoided. For the secondary bacterial infection large doses of bismuth and salines should be given. Astringents must very rarely be employed. High enemata are the most useful form of local treatment. Inject slowly 1 to 3 pints of silver nitrate (1 in 500) or eusol. For amœba carriers emetine periodide must be given in daily doses of 6 grains (0·02) for 12 days at a time. Two such courses will cure the majority of cases.

The patient complains of ACUTE DIARRHŒA, *coming on very suddenly, and attended with severe* COLLAPSE, *abdominal* CRAMPS, *and " rice-water " stools.* The disease is probably CHOLERA.

§ 245. IX. Cholera (synonym : Asiatic Cholera) is a disease, due to the comma bacillus of Koch, which commences with urgent vomiting, purging, and colourless evacuation, cramps and a tendency to collapse, and which, if not fatal in the first stage, is attended by secondary fever. The period of incubation is usually three to six days, but it may vary between one and ten.

There are three well-marked stages :

(a) *Stage of evacuation*, which lasts from two to twelve hours, or longer. The patient is *suddenly* seized with violent vomiting, severe cramp, and profuse diarrhœa. The stools, after the first few, are colourless and opaque, resembling rice-water, and containing flakes of columnar epithelium and casts of villi; and the *comma-shaped bacillus* (§ 667). There is severe cramp in the fingers, toes and abdominal muscles, great exhaustion, small and weak pulse, and coldness of the body. (b) The *algide stage*, cold stage, or stage of collapse, lasts a few hours to a few days according to the severity of the case. The patient becomes like a corpse; the surface temperature goes down, and the skin becomes a deadly livid hue; the pulse cannot be felt at the wrist. The temperature is most remarkable, for in the rectum it may be as high as 105° F., while in the axilla it is only 90° F. During this stage the purging ceases, but the vomiting and cramps persist. The mind remains clear. There is suppression of urine and bile. (c) *Stage of reaction.*—The pulse returns, the temperature rises, the bile reappears, the urine is scanty and deficient in urea. The temperature goes up, and may be attended by typhoid symptoms. The bowels are confined. There may be erythematous, urticarial and other eruptions upon the skin. This stage is followed by great weakness.

The *Diagnosis* is easy in severe cases on account of the extreme suddenness and severity of the symptoms. The copious colourless evacuations are characteristic of cholera. The only conditions which resemble it are acute poisoning by arsenic, croton oil, and other irritants, and certain cases of malignant malaria. The identification of the bacillus renders the diagnosis certain.

Etiology.—The disease occurs in great epidemics, but it fortunately has not visited this country, except sporadically, since 1865–6–7. Prior to that date there were epidemics in 1854, 1848, and 1832. In India it is endemic. As regards age, none are exempt. The season of the year in which all epidemics in this country have occurred has been the autumn and the end of the summer. The exciting cause is a specific organism, which must be introduced into the alimentary canal. As in enteric, the disease is communicated by the evacuations from the bowels and stomach, and requires the same preventive measures (§ 420 *et seq.*). The disease is usually communicated by drinking water which has become contaminated. But it may be conveyed in other ways, as by flies, through *want of cleanliness.* One attack does not give immunity from a second.

Prognosis.—It is a very serious disorder, and nearly all earlier cases of an epidemic are fatal. The average mortality was 60 per cent. Patients died in the algide stage, but the newer methods of treatment have reduced the mortality by half. In the reaction stage uræmic coma, hyperpyrexia, or the typhoid state may cause death.

Untoward Symptoms are blood in the evacuations, long stage of collapse, restlessness, extreme cyanosis, and absence of the pulse at wrist. Favourable signs are a perceptible pulse in the algide stage, the early occurrence of reaction, cessation of cramp, secretion of urine, and the occurrence of sleep. The commonest *Complications* are pneumonia, occurring in the reaction stage, bronchitis, pleurisy, parotitis, bed-sores, inflammation of the pharynx, genitals, or bladder, and corneal ulcers.

There are two rare *varieties* : (1) Choleraic diarrhœa, or " cholerine "—that is, cases like autumnal diarrhœa occurring during an epidemic of cholera. (2) Dry cholera, that is, where there has been no vomiting or diarrhœa, but all the other symptoms.

Treatment.—Prophylactic vaccine gives immunity for four months. Opium (ℳ xx. (1·2)) may be given at the onset of the preliminary diarrhœa, but never after the characteristic colourless evacuations have set in. Rest in bed, warmth, and fluid farinaceous diet are essential; animal albumens in soups and jellies are harmful. Observation of the specific gravity of the blood in the collapse of cholera, and of the failure of normal saline injections, led Sir Leonard Rogers to employ the hypertonic injections which have been so successful in the treatment of this dreaded disease. When collapse appears, saline injections by rectum are useful so long as the systolic blood pressure is above 70 millimetres ; below that point they are not absorbed, and an intravenous injection should be administered of sufficient amount (3 to 4 pints) to raise the blood-pressure to normal, and ensure excretion by the kidneys. The solution contains sod. chlor. gr. 120 (7·7), calc. chlor. gr. 4 (0·2), potass. chlor. gr. 6 (0·3) to a pint (600) of sterile water, and is given at the rate of 4 ounces per minute. Camphor, sips of tea and coffee, act as stimulants. Potassium permanganate gr. 2 (0·12) is given by the mouth every half-hour as an oxidising agent, which destroys the toxins of the cholera bacillus. A practical cholera outfit with full instructions was supplied to medical men with the army in the East (1916). In India, kaolin 7 oz. in 14 oz. water given at the outset of the disease often aborts it.

§ 246. Chronic Diarrhœa.- The term chronic diarrhœa signifies the occurrence of frequent *loose* evacuations, say three or more in the twenty-four hours, extending over a period of weeks, months, or even years (as in Sprue). It is usually, though not necessarily, attended by tenesmus. The stools should be examined (§ 239) whenever the cause is doubtful. In all intractable cases the anus should be carefully inspected. Tenesmus points to the presence of disease of the rectum.

I. Chronic Diarrhœa may be due to some of the same causes as **Acute Diarrhœa** (*q.v.*). In children tuberculous ulceration of the intestine, intussusception, worms, or bad feeding ; and in adults, errors in diet, ulceration, and chronic irritant poisoning, should be remembered.

II. **Fissure of the Anus**, slight ulcers or abrasions, or even an inflamed pile, may cause a chronic diarrhœa which long baffles investigation.

III. **Ulceration of some part of the Intestinal Canal** is perhaps the commonest cause of diarrhœa in England, and it will be well to mention here all the ulcerating lesions which may affect the intestine, in order from above downwards. (1) Simple ulcer of the duodenum is a rare condition which may arise from burns, or from the same causes as simple ulcer of the stomach (§ 231). There may be few or no symptoms till sudden peritonitis or copious hæmorrhage and melæna occur. (2) Ulcer of the lower part of the ileum may be due to tuberculosis or typhoid fever. (3) Ulcer of the cæcum may arise from the pressure of inspissated fæces

or some foreign body—*e.g.*, the bristle of a tooth-brush—which has been swallowed. (4) Ulcer of the vermiform appendix may similarly arise from foreign bodies or as part of appendicitis (*q.v.*). (5) Ulcer of the rectum is generally of malignant or syphilitic origin ; it is attended by the passage of blood and pus, and stricture may result. (6) Ulcers of the large intestine and rectum occur in the later stages of dysentery. These may contract on healing and produce stricture. (7) Cancer of the bowel may produce ulcer in any part of the bowel, but the most frequent situation is the sigmoid flexure. (8) Bright's disease, severe anæmia, and other wasting diseases. (9) To these some add catarrhal ulceration (§ 243, VII). (10) Ulceration may follow prolonged constipation with atony of the colon. (ii.) A submucous streptococcal infection may cause chronic diarrhœa with precipitate stools.

The commonest causes of ulceration in this country are COLITIS, TUBERCLE, SYPHILIS, CANCER, and in tropical climates DYSENTERY (§ 244).

1. COLITIS, inflammation of the colon, occurs in two forms, ulcerative (§ 253) VII) and mucous (see IV below) ; and causes one of the most intractable forms of chronic diarrhœa.

2. TUBERCULOSIS of the lungs may be attended by diarrhœa, even without ulceration of the bowel, and in such cases the diarrhœa is considered to be one of the symptoms of the hectic fever in pulmonary tuberculosis, or due to swallowing of the infected sputum. Tuberculous ulceration is recognised by (i.) evidences of tuberculosis in the lungs or other part of the body ; (ii.) the presence of night sweats and intermittent pyrexia ; (iii.) the stools are watery and bilious, and there is rarely any pain ; (iv.) The Tubercle Bacillus may be demonstrated in the stools by appropriate staining methods. Relief is generally effected by quinine and opium internally, combined with appropriate dietary ; if these fail recourse may be had to pernitrate of iron, opium, and lead.

3. INTESTINAL CANCER presents the following features: (i.) The patient is usually over forty-five or fifty, and there may be a family history of cancer ; (ii.) there may be cancer in the glands or other parts of the body, and there is almost always a history of *emaciation preceding the diarrhœa ;* (iii.) paroxysmal abdominal pains are frequent, and if the disease is in the rectum there is great pain and tenesmus on passing a motion ; if it be not in the rectum, a tumour can generally be made out through the abdominal wall ; (iv) the stools vary, but very often contain blood in considerable quantity.

4. In SYPHILITIC ULCERATION of the bowel (i.) the motions often consist largely of pus and blood ; (ii.) great pain and tenesmus are usual, combined with (iii.) other evidences and a history of syphilis. Stricture occurs in the later stages. (iv.) Opium and antisyphilitic treatment are here of great value to check the diarrhœa.

IV. Chronic Colitis or Mucous Colitis is in its early stages frequently overlooked, when the patients complain perhaps only of " nerves." The symptoms are (i.) attacks of diarrhœa alternating with constipation. During the attacks (ii.) mucus is found in the stools. The mucus may

be passed in masses, shreds, or casts several inches long. Occasionally blood is also passed, indicating ulceration. The fæces contain intestinal sand in one-tenth of the cases. (iii.) The general health is lowered, associated with despondency and other symptoms of nervous prostration, together with (iv.) abdominal discomfort and sometimes paroxysms of pain. (v.) Examination may reveal a distended and tender colon, particularly over the sigmoid; or spasmodic contraction of the descending colon may be felt. The sigmoidoscope is used as an aid to diagnosis in obscure cases.

Course.—The attacks last at first a few days only, then for weeks or months; the patient may at times pass nothing but mucus by the bowel. The disease is not of itself fatal, but is often very intractable to treatment. It leads to emaciation, neurasthenia, and, in the more serious cases, death from asthenia or complications. Relapses may continue for ten years or longer.

Treatment.—During the attacks rest, warmth, bismuth, kaolin and milk diet are essential. Between the attacks treatment is directed to prevent irritation of the mucous membrane by correct diet and prevention of accumulation of fæces in the colon. All seeds, skins, and stringy foods must be forbidden; examination of the fæces reveals that at some stages all fruits and vegetables are undigested, and should therefore be avoided. Combe, of Lausanne, found that nitrogenous foods favoured putrefaction, and he obtained successful results on a diet largely carbohydrate. Fruit juices, etc., are given to provide the vitamins which are lacking in a purely carbohydrate diet. The colon can be kept empty by paraffin oil, or other measures described under Constipation in § 252. Lavage of the colon daily, or twice weekly, with plain or medicated water, is now practised at Plombières, Harrogate, and other spas, and may be carried out at home under careful medical supervision. High injections of oil aid many cases and vaccines have assisted others. If the disease resist medical treatment, cæcostomy or appendicostomy may be performed, through which the colon is flushed out daily.

V. **Obstruction in the Portal Circulation** produces diarrhœa, due to the congestion of the intestinal wall. It is recognised by: (i.) A previous history of heart disease, or of intemperance and alcoholic dyspepsia; (ii.) other signs of liver or cardiac disease; (iii.) other evidences of portal obstruction, such as ascites, piles, and a large spleen (§ 284); (iv.) there is little or no pain, and the stools are liquid and dark, occasionally bloody. The *Treatment* requires caution, because the diarrhœa and hæmorrhage of themselves relieve the condition by diminishing the venous engorgement. (i.) If the diarrhœa has not lasted long, a large dose of calomel will relieve the portal congestion, and so cure the diarrhœa. (ii.) Magnesium sulphate, gr. 20 (1·2), with alum and dilute sulphuric acid, are recommended; bismuth and opium, with caution, are the most useful for checking the diarrhœa.

VI. **Dysenteric Diarrhœa** is a sequel of dysentery, which may perhaps have been contracted abroad many years previously. The laity, seeking a more elegant term, often speak of any form of diarrhœa as " dysentery." The characteristic symptoms here are: (i.) A previous history of acute dysentery, or a residence in dysenteric countries; (ii.) the tongue is generally characteristic, being very clean, red, and often

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sore; (iii.) the stools vary, but are generally pale, pasty, frothy, and easily ferment; (iv.) slight errors in diet produce great aggravation of the diarrhœa. The *Treatment* consists almost entirely in regulating the diet. Only milk, farinaceous food, and eggs should be allowed; no meat, vegetables, or fruit. In severe cases the patient should live on boiled skimmed milk, 4 or more pints a day. Rest and warmth are very important. Bismuth, with or without opium, checks the diarrhœa. Daily colonic irrigation with arg. nit. (1 in 1000), or mild antiseptics, is useful.

VII. **Nervous Diarrhœa** is a form of diarrhœa which may continue for years; it occurs in nervous people and has the following characteristics: (i.) The motions are often quite healthy, sometimes liquid, never attended by melæna or mucus. There is usually no pain or tenesmus. The diarrhœa is generally recurring or intermittent, occurring in the early morning, or when the patient is "nervous." Sometimes it follows each meal (*lienteric* diarrhœa). (ii.) It occurs for the most part in females of a neurotic type. (iii.) Diet seems to produce little or no influence, but the *attacks are determined* by mental emotion or bodily fatigue. A plain but generous diet is called for; and the administration of nux vomica, belladonna, and bromides is often more efficacious than astringents. Careful search should be made for any source of uterine or other reflex irritation. Arsenic (♏ ii. (0·12) Fowler's solution), with meals, is said to be a specific for lienteric diarrhœa.

The crisis of LOCOMOTOR ATAXY sometimes takes the form of acute diarrhœa, with or without pain. In HYSTERIA acute attacks of diarrhœa, with noisy borborygmi, may occur, determined in the same way as other hysterical attacks.

VIII. **Amyloid Disease** of the intestines gives rise to a most intractable form of chronic diarrhœa. Indeed, this is the common mode of death in amyloid disease of the viscera. The characteristics here are: (i.) A history of long-standing purulent discharge, or of syphilis; (ii.) great pallor of the skin, accompanied by evidences of lardaceous disease in the spleen, liver, and kidney; (iii.) the stools are generally liquid and extremely offensive, sometimes attended by hæmorrhage. The *Treatment* is very unsatisfactory. Pernitrate of iron, sulphuric acid, logwood, acetate of lead, may be tried, and also opium, which does no harm, even when there is amyloid disease of the kidney, as there is no tendency to uræmia.

The *rarer* cases of chronic diarrhœa are:

IX. **Senile Diarrhœa** was, I believe, first described by Maclachlan, in his "Diseases of Old People." It occurs in persons over sixty or seventy, and is very chronic in its course, but the patient suffers very little. Careful examination for organic disease should be made before concluding that the condition is simple senile diarrhœa. Astringents and most other remedies fail to relieve it, and it may exist for many years without emaciation or danger to life.

X. **Mineral Poisons**, and especially arsenic and antimony, in small continuous doses, may cause persistent diarrhœa. It was in this way that the celebrated Maybrick case was discovered.

XI. **Pancreatic Disease** has been associated with diarrhœa. Dr. Burney Yeo has described a case of chronic diarrhœa which resisted all treatment until pancreatic emulsion was administered. The diarrhœa returned when this was stopped, and ceased again on its administration. It may be assumed that only the chronic forms of pancreatic disease (*e.g.*, Fibroid Pancreatitis) would be attended by this symptom, and the diarrhœa is probably dependent upon the excess of fat and undigested muscle fibre in the fæces (§ 205).

§ 247. XII. **Psilosis or Sprue** is a condition met with in the tropics. It is characterised by diarrhœa and other symptoms of congestion of the alimentary canal usually running a prolonged, and often fatal, course.

The *Symptoms* consist of (i.) diarrhœa, which is very chronic and continuous, and attended by pale, copious, and frothy stools, which are mostly passed in the early hours of the day and are acid in reaction; (ii.) dyspepsia, with distension of the abdomen and emaciation; and (iii.) tenderness of the mouth. At times these symptoms are exacerbated, and aphthous patches appear on the mucous membrane of the

mouth and pharynx. In the course of time the patient becomes extremely feeble. Women are more often affected than men. If untreated, the disease is usually fatal in one or two years; even with treatment it may lead to death in six or ten years. Much depends upon the age of the patient; in late middle-age cure is unlikely.

The *Treatment* consists in giving as much rest as possible to the alimentary canal. The patient must be put to bed, and kept on a restricted milk diet for six weeks or more. Only small quantities must be given at a time. The patient may gradually return in the course of a few months to ordinary diet, but meat and coarse vegetables must be taken only seldom, even after recovery. When milk disagrees, after trying condensed and peptonised milk, raw meat juice may be given for a time. It is important not to give large quantities per diem, however much the patient may complain of hunger. Begin with only 2 pints of milk daily. This may be increased, when the mouth is not tender, to 5 or 6 pints a day, in the course of two months. The juice of raw fruits, especially strawberries, has been recommended. Alcohol is injurious. Good results are reported from vaccine therapy, with organisms of a streptococcal type. Scott introduced treatment with parathyroid extract gr. ₁⁄₆ twice a day and calcium chloridegr. x. three times a day; the results are most encouraging.

§ 248: **Tenesmus literally means straining** at stool (τείνω, to strain or stretch); but in its widest sense it may be taken to mean any local rectal sensation of "bearing down" which results either in a constant desire to go to stool, or a straining when at stool. The latter may lead to prolapse of the rectum, especially in children. Diarrhœa is always attended by more or less tenesmus, but tenesmus is not always attended by diarrhœa. (1) Ascertain if the tenesmus is accompanied by diarrhœa – i.e., are the motions frequent and liquid? If so, refer to the section on Diarrhœa, § 242. (2) Particular attention should also be paid to the shape and consistence of the motions. (3) Examine locally for any anal or rectal condition such as fissures, piles, polypi, or ulcers. All the pelvic organs should also be very thoroughly investigated, especially in women, in whom the symptom is commoner than in men

Causes.—Tenesmus (not necessarily accompanied by diarrhœa) may arise from four groups of causes:

1. Various conditions of the ANUS—pruritus, eczema, or fissure—may be overlooked for a long time. Piles also, if internal, may be difficult to detect, even by the examining finger, but streaks of bright blood will appear in the motions from time to time.

2. Various RECTAL CONDITIONS, especially stricture or ulceration. The former (usually of syphilitic origin) is attended by tape-like stools; the latter is attended by pus or blood, or both. Prolonged use of purgatives, or the constant use of the glycerine enema may result in straining at stool and prolapse of the rectum. Proctitis (inflammation of the rectum and anus) is another cause. In the aged, we should always suspect cancer of the rectum, although this is usually attended by actual diarrhœa.

3. PRESSURE on, or irritation of, THE RECTUM FROM WITHOUT, such as may be caused by chronic congestion, version, or other disease of the uterus. These in women, and congestion or new growth of the prostate, in men, are both very common causes. Any bladder disease, such as stone—a frequent cause of tenesmus in children, and apt to result in

prolapse of the rectum—or new growths or chronic cystitis may cause this distressing condition. Ischio-rectal abscess, pelvic hæmatocele, and various ovarian and Fallopian tube lesions are all apt to cause tenesmus. The catamenial period may be attended by a certain amount of tenesmus.

4. In HYSTERICAL AND NERVOUS SUBJECTS any fright or other emotion may at once determine tenesmus, which is spoken of by the patient as "diarrhœa." In tabes dorsalis the "rectal crises" may take the form of tenesmus.

Treatment.—The indications are (1) the removal of the cause, the treatment of piles and other causal conditions being found elsewhere; (2) the relief of any local congestion or irritation of the rectum. Fissure may require antisyphilitic remedies. In any case, morphia, belladonna, or hydrochlorate of cocaine in the form either of suppositories or enemata will relieve the distress from which the patient suffers.

§ 249. **Blood in the Stools** is met with, as we have seen, in dysentery and some cases of simple diarrhœa; but it may be met with unassociated with the latter. The presence of blood in the stools may be recognised by the reddening of the water in which the stool is placed, or by the spectroscope. Clinically, blood in the stools may present two widely different characters : (*a*) When the blood is of *bright crimson colour* it indicates either that the bleeding comes from the rectum or the lower part of the large bowel; or, if it comes from the upper part of the intestinal canal, that it is too large in amount to be acted upon by the intestinal secretion. (*b*) *Melœna (tar-coloured stools)* is met with when hæmorrhage in moderate quantity has taken place in the stomach or the upper part of the alimentary tract, in which case the digestive fluids of the stomach and intestine acting on the blood give it a tarry colour. The causes of these two conditions may to some extent be interchangeable, for what will produce a large hæmorrhage at one time may at another produce only a little.

(*a*) **Bright Red Blood** may be due to the lesions of the lower part of the alimentary canal. Of these, 1, 2, 3, 5, and 7 are referable to the anus or rectum, and may generally be discovered on local examination.

1. HÆMORRHOIDS, or PILES, are undoubtedly the commonest cause of blood in the stools. The blood is generally met with in streaks only, but the quantity may at other times be very large. This condition is fully described below, § 250.

2. FISSURE OF THE ANUS may also produce streaks of blood. It is a not infrequent condition, and is recognised by the excruciating pain during and after defæcation. The irritation it causes may give rise to a variety of false diarrhœa. The fissure can always be seen by *careful* examination.

3. RECTAL ULCERS may give rise to streaks of bright blood in greater or less quantity, mixed with pus and mucus. They are usually of syphi-

litic, cancerous, or dysenteric origin, and can frequently be felt by digital examination (§ 246).

4 A discharge of blood-stained mucus, coming on somewhat suddenly in an infant, is highly suggestive of INTESTINAL INTUSSUSCEPTION, which is one of the causes of acute obstruction (§ 253).

5 RECTAL POLYPI are met with chiefly in children.

6 TYPHOID and TUBERCULOUS ULCERATION of the small intestine
› sometimes produce very profuse discharges of bright red blood, which comes from the lower end of the small intestine. Other evidences of these affections are present.

7 SCHISTOSOMA MANSONI, a distinct species of trematode, discharges its ova in the veins about the lower end of the bowel. The ovum is armed with a lateral spine and the irritation which it sets up causes a spurious dysentery and the formation of polypoid masses within the rectum. Schistosoma hæmatobium may also affect the bowel (§ 300). The ova are characteristic (Fig 89). *Schistosoma japonicum*, a third species, gives rise to Katayama Disease or Schistosomiasis of the Far East The ova show a knob-like projection at one side.

8. Various GENERAL BLOOD CONDITIONS may give rise to hæmorrhage coming from the rectum or elsewhere in the alimentary canal in varying amount. This occurs in purpura, scurvy, hæmorrhagic forms of the specific fevers, acute yellow atrophy of the liver, and leukæmia

(b) Melæna (*tarry stools*) is met with when bleeding takes place in moderate quantity from the stomach, or high up in the alimentary tract. Its causes are :

1. When coming FROM THE STOMACH, it may be associated with profuse *hæmatemesis* (§ 216), the commonest causes of hæmatemesis are gastric ulcer and hepatic cirrhosis. Duodenal ulcer must be borne in mind.

2. PORTAL OBSTRUCTION (§ 258) is one of the most frequent causes of melæna, especially that form due to alcoholic cirrhosis of the atrophic variety. It may also occur with advanced cardiac disease In either case the hæmorrhage in these circumstances is a natural safety-valve, and gives relief to the engorged state of the portal circulation

3. CANCEROUS, TUBERCULOUS, and other ULCERATIONS of the small intestine (see §§ 243 and 246), and lardaceous disease of the bowel may also produce melæna. Colitis may cause traces of occult blood

4. The GENERAL BLOOD CONDITIONS above named, when the hæmorrhage is small in amount, are attended by tarry instead of bright red stools. *Melæna neonatorum* is a rare condition in which there is a passage of blood in new-born children. Nothing is known as to its cause. It may prove fatal or disappear, leaving the child anæmic but otherwise none the worse.

5. The ANKYLOSTOMUM DUODENALE (ankylostomiasis) is a frequent cause of profuse melæna in Egypt and other foreign countries (§§ 239, 441).

The *Treatment* of melæna should be directed to the cause, but the general principles are those laid down for hæmatemesis (§ 216), Tur-

pentine (10 minims capsule), lead acetate, and opium are recommended. Suprarenal gland has recently been advocated as a remedy. *Ankylostomum* is readily destroyed by thymol (§ 441). Until recently melæna neonatorum defied all treatment, but many cases have now been reported in which the subcutaneous injection of serum has stopped the hæmorrhage. Human serum is best, but if not available fresh horse serum or antidiphtheritic serum may be used. The initial dose is 5 c.c., but this may have to be repeated until 60 to 70 c.c. have been given.

§ 250. **Hæmorrhoids**, or Piles, consist of a varicose condition of the rectal veins. This varicosity forms a swelling of variable size, which may be altogether within the anus (internal piles), or partly internal and partly external. Internal piles may in some cases be seen, when the patient " bears down," as small purple swellings just protruding from the sphincter; in other cases internal piles are discovered only on digital examination of the rectum.

Symptoms.—(1) Streaks of *bright red* blood occur in the stools, and sometimes as much as ¼ pint of blood may be passed at one time. (2) There is pain on defæcation, the pain continuing for some time after the passage of a stool. When a pile becomes inflamed, or strangulated by the sphincter, severe pain and discomfort is experienced, and the patient may have to remain in bed for days. Pain may be referred to other parts of the body—*e.g.*, to the testicles, bladder, or loins. (3) Constipation nearly always accompanies piles, due partly to mechanical obstruction, and partly to the pain caused by defæcation. (4) In severe cases constitutional symptoms are developed, such as lassitude, irritability, headache, faintness, and later on anæmia, from loss of blood.

Etiology.—(1) Portal obstruction is itself a cause of piles, and in all marked cases we should seek for the other symptoms of this lesion (§ 258). (2) Habitual constipation, however, is undoubtedly the most common cause of hæmorrhoids, particularly in women, who in early life are so apt to contract this habit. (3) Alcohol, especially in the form of malt liquors, with excess of sugar, causes portal congestion, and thus becomes a source of piles. Alcohol in any form aggravates the condition. (4) Sedentary occupations and deficient exercise also produce piles. (5) Various local conditions, such as sitting on soft cushions which constrict the inferior hæmorrhoidal veins, uterine displacements, pelvic and other tumours, are all potent causes of hæmorrhoids.

Prognosis.—Hæmorrhoids are not usually regarded as serious, but they may be extremely troublesome, partly by the constant loss of blood, partly by their liability to repeated attacks of inflammation, and partly by the pain they cause.

Treatment.—Much may be done by three simple means: (1) The avoidance of alcohol (especially malt liquors) and sugar; (2) keeping the piles scrupulously clean; and (3) the bowels regularly and loosely open. Prolapsed piles must be replaced at once. Rich food, wines and other causes of hepatic congestion must be forbidden. Confect. sulph., or sennæ,

with an occasional cholagogue at night are good ; paraffin is apt to cause the piles to descend. Local applications should be of the simplest kind. The old-fashioned gall and opium ointment is now very properly replaced by hamamelis, with conium, morphia, or cocaine for the pain if necessary. Liquid hazeline is an excellent preparation, and is best applied on a strip of lint inserted within the anus, and left there ; or a suppository may be employed, containing gr. 1 to 3 (0·06–0·2) of hamamelin, and
• morphia gr. ⅛ (0 007) if requisite. Inflamed piles are very painful, and arc best treated by warm hip-baths, frequent bathing, sitting over hot water in a bidet, warm fomentations with opium, belladonna or cocaine. Removal by surgical measures is called for in some cases, but many cures can be obtained by high frequency and diathermy when administered locally by experts.

§ 251. **Intestinal Worms** [1] may give rise to no symptoms at all. They are most frequently met with in children, and may remain undiscovered until they are found in the stools. The morphology, symptoms, and habitat of the various entozoa are described in Table XVII, p. 342. Threadworms (Fig. 64) and roundworms (Fig. 65) are the most common. It used to be considered that threadworms lived in the colon, but it is now believed they exist in the caput cæcum, and sometimes in the vermiform appendix. This fact explains those cases which appear to be cured for a time, but which continually relapse.

The *Symptoms* are very definite, and consist of : (1) Vague and persistent, though often paroxysmal, pains in the abdomen ; (2) capricious and sometimes ravenous appetite, in spite of which the child becomes thin and sallow ; (3) grinding of the teeth at night, picking of the nose, and other reflex phenomena , (4) irregularity of the bowels, or diarrhœa. Threadworms produce intense itching of the anus, and consequently fidgettiness, especially at night. They may wander forwards and cause vulvitis. In addition to the above symptoms, they may give rise to nervous signs so severe as to suggest meningitis. Ankylostomum, tricocephalus, bilharzia, and distoma cause severe anæmia and debility, and are described under anæmia (§ 441).

The *Prognosis* is usually good when the cause is discovered. Occasionally round worms have led to death from perforation of the bowel ; and they have been found in the gall-bladder, Eustachian tube, and bronchi.

The *Treatment* differs for the different worms. For *threadworms* the best treatment consists of quassia injections. After an aperient, ℥ i (32) of powdered quassia to a pint (600) of boiling water is, when cold, injected slowly into the bowel, and retained as long as possible. Common salt injections of the same strength may be used nightly. The worms are destroyed with two or three such injections. Santonin, gr. i (0·06), in a powder with calomel is very efficacious ; it should be given on alternate days for three doses, followed by castor oil. Where the worm has its habitat high up in the intestine (as the *tapeworm*), treatment is conducted in three stages. (1) In order to starve the parasite by keeping the alimentary canal as empty as possible, the patient should have no food after midday, and at night or the next morning a purgative must be taken. This leaves the worm uncovered, and thus readily acted upon by (2) the anthelmintic, which is given about an hour after purgation. The chief anthelmintic is ext. filicis liq., ℥ i (4). Some recommend ♏ 30 (2) of spirits of turpentine to be given with this ; others give kousso ℨ iv (16) or pelletierine (2 gr. (0·12) of the alkaloid). (3) Two hours later give calomel with a saline aperient, to eject the worm from the body. The stools must be examined to see that the head is passed. If only segments are passed the worm will grow again, and the same treatment will have to be repeated within three months. For the *round worm* the specific remedy

[1] Intestinal myiasis is rare in this country. The " worms " are the larvæ of flies.

is santonin, given in 2 grain (0·12) doses to a child of three and upwards; for an adult gr. 5 (0·3) are given. Follow with a purge. For *Ankylostomum Duodenale*, see § 441.

§ 252. Constipation is insufficient action of the bowels, delay in the passage of the contents of the intestine, causing hard, dry fæces (scybala), independent of organic disease within or outside the intestinal canal. This source of fallacy must be carefully excluded before diagnosing a case as one of simple constipation. The delay is usually in the colon, the pelvic colon or the rectum. A useful and simple test consists in giving a tablespoonful of powdered charcoal at night; it should normally have completely disappeared from the stools before forty-eight hours.

The *Symptoms* which accompany or result from constipation are sufficiently familiar—at first headache, languor, and depression, followed by a furred, coated tongue, dyspepsia, sallow or pigmented skin, anæmia, sleeplessness, and eruptions, usually of an urticarial or erythematous nature. The temperature may rise a degree or so in certain conditions from temporary constipation, and I have met with cases where it went up to 102° F. The retention of hard fæcal masses may give rise to an alternating diarrhœa, which leads to error in diagnosis. Hæmorrhoids result from habitual constipation, and a distended ulcerated colon or atony of the colon. In women, in whom the condition is more common than in men, chronic constipation may even lead to uterine disease. In both sexes varicose veins, œdema of the legs, sciatica, especially on the left side, and numbness of the legs may occur. In some cases there may ensue ptosis of part of the intestine with formation of the bends or " kinks " which have been described by Sir Arbuthnot Lane. This leads to delay of the bowel contents; then follows prolonged ill-health due to the toxæmia of INTESTINAL STASIS.

For purposes of treatment we may consider the Causes of simple or uncomplicated cases of constipation under three headings:

(a) **Errors of Diet.**
  (i.) Too bland food—*e.g.*, no vegetables, no food with coarse residue.
  (ii.) Too dry food—*e.g.*, deficient fluid ingesta.
  (iii.) Too little or poor food, deficiency of vitamins.

(b) **Causes of Defective Peristalsis**, other than errors of diet.
  (i.) Sedentary habits.
  (ii.) Depressing emotions, anxiety, worry, etc., cause sympathetic inhibition, hence spasm.
  (iii.) Old age and other conditions with poor general tone, such as anæmia.
  (iv.) Prolonged disregard of the calls of nature, with dilatation of rectum and pelvic colon consequent on blunted sensation.
  (v.) Weak abdominal muscles.
  (vi.) Atony of the colon, with or without colitis.
  (vii.) Some febrile states.
  (viii.) Endocrine disorder, specially deficient activity of thyroid and pituitary.
  (ix.) Reflex spasm, as in catarrh of the colon or uterine disease.
  (x.) Disease of brain or cord, such as tabes and cerebral tumour.
  (xi.) Drugs, such as opium, iron, lead.

(c) Deficiency of Bile, or Intestinal Secretions.

   (i.) Functional inactivity of the liver.
   (ii.) Profuse vomiting
   (iii.) Excessive loss of fluid by skin or kidneys.
   (iv) Astringents, such as chalk or catechu. Hard waters also act in the usual way.

Treatment.—Chronic constipation is serious in respect of the trouble-some consequences mentioned above. For correct treatment we must first *find out the cause.* With the patient lying down and the muscles well relaxed examine the colon to see if it be distended or loaded ; place one hand at the back, and press it forwards between the iliac crest and the last rib to meet the other hand, which is placed flat on the anterior abdominal wall. An X-ray examination assists in deciding the presence or absence of mechanical obstruction, and the position of chief delay in the passage of the intestinal contents (§ 222). In cases presenting profound general toxæmia one or several of Lane's " kinks " may be revealed, and from the X-ray findings it can be decided whether surgical or medical measures should be adopted. Having excluded local causes by a thorough examination, we should consider the various causes above mentioned. The treatment of constipation comes under six headings.

  Dietetic Treatment.—Increase the amount of fluid taken— *e g.*, by sipping a tumbler of cold water slowly whilst dressing in the morning and undressing at night. Avoid large quantities of milk or hard water. Coarse foods should be eaten, such as oatmeal, wholemeal, or brown bread, green and raw vegetables, onions, figs, prunes, and ripe fruits (see § 236). A teaspoonful or tablespoonful of salad-oil at meal-times is often very efficacious. Various liquid paraffin preparations act as lubricants, assisting the passage of hard intestinal contents. Inculcate regular *habits,* even when there is no inclination to go to stool. Psychotherapy is useful in this connection. Active *exercise* is advisable except when uterine or ovarian disease is present. A systematic exercise may be practised by lying flat upon the back and rising from a recumbent position without the aid of the arms ten or a dozen times each morning or evening. Many exercises are now taught which strengthen the abdominal muscles. Electricity is used in various ways , some forms relax spasm, others stimulate to healthier muscular action. Abdominal *massage* is useful. Gently " rolling " the abdominal wall, or rolling a 7-pound shot-ball over the abdomen in the direction of the hands of the clock   (5) Where signs of deficient action of the thyroid or pituitary are present, extracts of these glands greatly aid constipation. Bile extracts are very efficacious in other cases. *Drugs.*—For occasional constipation, aloes with the evening meal and a seidlitz-powder in the morning are the most harmless. Calomel or other mercurial preparations should not be given habitually, but may be taken once a week for a few weeks. Aloes combined with phenolphthalein is an excellent preparation, particularly where the colon is at fault. Cascara, aloes and senna are the best drugs for frequent use. A

useful vegetable pill is pil. col. co., pil. rhei co., āā gr. j. (0·06), ext. hyoscyami, gr. ½ (0 03) ; two at bedtime. Another good formula is Tr. Nuc. Vom., Tr. Belladonna, āā ℞ v. (0·3) ; Tr. Hyoscyam. ℞ x. (0·6), Ext. Casc. Sag. liq. ad ʒi. (4). Belladonna and nux vomica in small doses undoubtedly promote peristalsis ; the former is especially useful to relax spasm as in conditions of pelvic irritation. Jalap, elaterium, scammony and gamboge are useful when drastic purgation is desired. Salines given daily for some weeks will often re-establish the functions of a torpid intestine (F. 46). These may be given in the form of the mineral waters, such as Carlsbad, which contains 13 grains of sulphate of soda to the tumbler, with alkalies (dose, one or two tumblers twice daily) ; Friedrichshall, which contains 60 grains of the sulphates of magnesia and soda with alkalies (dose, half a tumblerful daily) ; Hunyadi water, which contains 200 grains of sulphates of soda and magnesia with alkalies (dose, quarter to half a tumbler). All of these are best given on an empty stomach (F. 57, 88, and 90 are also useful). An excellent aperient for children is cascara and maltine mixed together in the proportion of ℞ 10 to 20 (0 6–1·2) of the ext. casc. sagrad. liq. to the teaspoonful of maltine. *Enemata* are useful in conditions of atony of the descending and pelvic colon, and pouched rectum , it must be remembered that they do not empty the small intestine. The ordinary soap enema of 1 or 2 pints of soapy water may be used. Half an ounce of glycerine is a very effective enema, but it should not be used longer than a few weeks, for it produces an irritable condition of the rectum. In cases of very prolonged constipation ¼ to ½ pint of olive oil may be given every night. If this be injected very slowly, it is retained, and after a course of one or two weeks it is wonderful how regularly the bowel resumes its functions.

Colon irrigation with normal saline is often effective and necessary, particularly where hard masses can be felt in the cæcum. Two or three pints at body temperature are introduced slowly under pressure, and are immediately evacuated. Carried out daily for a week, then on alternate days, and ultimately once a week, this is very effective in clearing the colon of accumulated fæces. Gradually the bowel resumes its normal functioning. The only type of case in which this is not very satisfactory is that in which there is considerable ptosis and as a result the whole of the saline is not evacuated at once. The repeated calls to stool are annoying and frequently this type of case complains of depression and increase of symptoms, probably due to the more easy absorption of toxins dissolved in the retained saline.

Hirschsprung's Disease is a condition of atony and dilatation of the colon of congenital origin. The cause is unknown.

Symptoms.—There is obstinate constipation, starting in the first weeks of life, and subsequently tympanites with visible peristalsis, auto-intoxication and emaciation. If early childhood is survived, complications such as peritonitis, volvulus and intestinal obstruction may ensue. The disease is fatal in the absence of treatment.

The *Diagnosis* can only be made by the history and obvious signs of a distended colon. A similar condition may be acquired by prolonged bad habits, but this is not Hirschsprung's disease.

Treatment.—Attend to the diet and stimulate the intestinal muscle with strychnine, massage and electricity. Large enemata may be tried. Operation may be necessary— " short-circuiting " by Lane's method, with or without extirpation of the large bowel.

The patient complains of SUDDEN STOPPAGE OF THE BOWELS *with inability* to pass even flatus, ABDOMINAL PAIN, *and* VOMITING *which gradually becomes stercoraceous ; his* PULSE IS RAPID, *and there is a tendency to* COLLAPSE.   The case is probably one of ACUTE INTESTINAL OBSTRUCTION.

§ 252. **Acute Intestinal Obstruction** is one of the most serious medical or surgical emergencies to which a medical man can be summoned.

The *symptoms common to all forms* of acute obstruction are (1) complete • *constipation, not even flatus being passed.*   (2) The *pain* is at first paroxysmal, referred to the umbilicus, though it becomes continuous later on. There is not usually much tenderness.   (3) The *vomiting* comes on earlier, is more urgent, and becomes more rapidly stercoraceous in proportion as the obstruction has taken place high up in the intestines.   (4) Abdominal distension is generally present, and this may be one-sided, so giving us a clue to the position of the obstruction.   (5) Constitutional symptoms gradually supervene, with prostration and a thready, *rapid pulse.*   These also are more urgent when the small intestine is involved.   The urine is diminished in proportion as the obstruction is near the stomach, for then the vomiting is more urgent.

Diagnosis of Acute Intestinal Obstruction.—When summoned to a case presenting these three symptoms—stoppage of the bowels, acute abdominal pain, and vomiting—the first step is to identify the case as one of acute obstruction.   In *colic* (renal, hepatic, or intestinal) all of these three symptoms may be present, but the patient's general condition is not so serious.   Moreover, the position of the pain in renal and hepatic colic is characteristic (see § 196).   In *acute peritonitis* there is great tenderness over the abdomen, thoracic respiration, and some fever (see also § 194). But when there is *perforation* into the *peritoneum* collapse is present, at first without fever, and perforation is diagnosed with difficulty only by (i.) the passage of wind by the bowel ; (ii.) the collapse being much greater even than that in acute obstruction ; and (iii.) a possible history of the condition which has resulted in perforation or rupture (consult also § 193). It is sometimes impossible to diagnose these two conditions, and an exploratory operation should be undertaken without delay.

Causes of Intestinal Obstruction.—It is of some importance to ascertain the cause, for the prognosis and treatment differ somewhat in each case. (a) In *acute* intestinal obstruction, in which the symptoms come on *suddenly* in a person previously healthy, there are three *common* causes : (1) External hernia ; (II) intussusception ; (III) internal strangulation. (b) Sometimes, however, acute will supervene on chronic obstruction, and the *common causes of chronic obstruction* (§ 254) are four in number : (I) Malignant stricture of the bowel ; (II) simple stricture ; (III) pressure of a tumour ; and (IV) dilatation of the bowel.

Features special to the several causes of acute intestinal obstruction.

I. EXTERNAL HERNIA is known by the presence of a tumour in the femoral, inguinal, or umbilical region.   No impulse on coughing is present. Obturator hernia is very rare, and is usually only discovered at the time of operation.

II. INTUSSUSCEPTION, or invagination of the bowel, is by far the commonest cause in childhood. According to Brunton, it is a cause of 43 per cent. of all cases of obstruction. True intussusception is always from the bowel above into the part below, and in more than half of the cases the lower part of the ileum becomes invaginated into the cæcum. In a third of the cases some other part of the ileum, and in about one-eighth some part of the colon, is implicated. The invaginated portion slowly sloughs, the two edges may be welded together, the slough may • pass about the eighth or tenth day; thus spontaneous recovery may occur, though this is relatively rare. Death from perforation and collapse is more usual unless the case is dealt with surgically. Intussusception is known by (i.) severe tenesmus; (ii.) a rectal discharge of *blood and mucus* with a red jelly appearance; (iii.) a sausage-shaped tumour may be felt, altering in position, on palpating the abdomen, and in extreme cases the invaginated portion of bowel is felt *per rectum ;* and (iv.) the patient is a child, usually under two years of age.

III. INTERNAL HERNIA or STRANGULATION—*e.g.*, by bands of adhesion —is known by (i.) the urgency of the symptoms; (ii.) the patient is an adult man, with (iii.) a history of old peritonitis. VOLVULUS (or twisting of the bowel) may be indistinguishable from the preceding—indeed, it practically results in strangulation—but (i.) it occurs in men over forty, usually with a history of chronic constipation; (ii.) abdominal distension may be great; (iii.) sometimes a tumour is felt over the sigmoid flexure, the usual site of volvulus.

Internal strangulation may also arise from (1) adhesion of the end of the appendix vermiformis through which a knuckle of the bowel gets nipped. (2) Adhesions of the bowel. This is a cause of 3½ per cent. of the cases of acute obstruction. (3) Congenital deficiencies in the mesentery or bowel, or the foramen of Winslow.[1]

The rarer causes of acute obstruction are three in number :

IV. IMPACTION IN THE BOWEL of a large GALL-STONE. This is not so rare as might be supposed, and Dr. Murchison was able, without much difficulty, he stated, to collect thirty-four cases. A large gall-stone escapes from the gall-bladder by ulceration into the bowel. The obstruction is high up in the small intestine, and consequently (1) the pain and constitutional symptoms are of extreme severity, and of very sudden onset. (2) The patient is usually a female (four females to one male), at or beyond middle age. (3) There may be a history of biliary colic, and in all cases there is a history of localised peritonitis some weeks or months before the seizure. (4) The symptoms may intermit, from the stone shifting its position.

V. Obstruction of the bowel may sometimes be due to an EXTRAVASATION OF BLOOD into the coats of the intestine. It occurs only in purpura, hæmophilia, and other blood disorders. Such cases are recognised by evidences of hæmorrhage, in other positions—melæna, epistaxis, purpura, or a history of urticaria or angioneurotic œdema.

VI. Among the still rarer causes of obstruction may be mentioned masses of

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[1] The rarer conditions are connected with congenital malformations. For instance, an interesting case of a patent Meckel's diverticulum into which the posterior wall of the ileum became intussuscepted, forming an umbilical tumour, in a male child, æt. six weeks, is published by Dr. Leonard Guthrie in *Pediatrics,* July 1, 1896, vol. ii.

round worms (Trousseau), impaction of too much cellulose, orange-peel, etc., hair-balls, concretions of ammonio-phosphate of magnesium (a frequent cause in horses, though rare in man), and other foreign bodies in the intestine.

Clinical Investigation and Diagnosis of the Cause of Obstruction.—If the case occur in a child, and there is a history of diarrhœa for the past few days, it is almost certainly intussusception ; in an old person suspect rectal stricture, impacted fæces, or volvulus ; in a young adult suspect strangulation or hernia. If the vomiting come on early and is urgent, it points to a tight constriction *high up* in the intestinal tube. So also after the onset of obstruction high up there may be a movement of the bowels. If the distension is chiefly in the centre of the abdomen, the obstruction is probably above the ileo-cæcal valve ; if it is chiefly in the flanks, the obstruction is below the valve ; if more in the right than in the left flank, the obstruction is probably in the splenic flexure.

When called to such a case, first examine for swelling in the positions of external herniæ. If the abdomen be distended, and present visible waves of peristalsis, inquire as to the causes of chronic obstruction (*infra*), as the case is probably an acute supervening upon a chronic obstruction. Always *examine per rectum*, for in acute intussusception the invaginated part of the bowel may be felt *per rectum*, and there may be a discharge of blood and mucus ; or a stricture or other cause of chronic obstruction may thus be discovered. Next inquire into the past history—*e.g.*, for peritonitis (as this is a cause of internal strangulation), or for appendicitis or hepatic colic. Then examine the abdomen by palpation and percussion for tumour or tenderness. If the abdomen is distended only on one side, the site of the obstruction may be localised.

Prognosis.—The prognosis of obstruction of the bowels is always very serious. Death occurs in the natural course either from (1) gangrene and rupture of the bowel, or (2) exhaustion and collapse. At the present day the prognosis almost entirely depends upon the *stage at which the case comes under notice*, and the treatment adopted. All the acute cases require early surgical interference, and a surgeon should be summoned at once. The success and justification of such diagnostic operations form one of the chief triumphs of modern surgery. As regards the *Causes*, obstruction from a gall-stone is perhaps the most serious, then intus-susception, then internal strangulation. Among the gradual causes, car-cinoma of the bowel gives the gravest prognosis, and paralysis the most favourable. Cases in which the obstruction is high up are less favourable than those in the large bowel.

Treatment.—Acute intestinal obstruction is one of those serious con-ditions that demand the resources of both a physician and a surgeon, who should jointly undertake the management of a case. The indications are (1) to ascertain the cause ; (2) to endeavour to remove the obstruction ; and (3) in the meantime to support the strength and relieve the pain by controlling the peristalsis upon which it depends. Enemata may be given in all cases ; purgatives should be avoided. Warmth is applied

to the abdomen in the form of hot fomentations, turpentine, belladonna, or opium stupes. If there are signs of peritonitis, cold is said to be more efficacious. The question of the administration of opium is debated (see Appendicitis), but, generally speaking, for the relief of the pain opium may be given as soon as the diagnosis is certain. The diet should consist of fluids, such as iced milk, beef-tea, and stimulants, given in small quantities, and frequently.

In *external hernia*, after a warm bath, it is best to proceed at once to operation. In *intussusception* some mild cases have a tendency to spontaneous recovery. Some surgeons recommend that an attempt should be made to reduce it by injections of warm saline or olive oil, but it is better to proceed at once to laparotomy. In *internal strangulation* or twisting it is best to operate without delay if an injection does not relieve and we are certain of the diagnosis. In cases of recovery without operation there has probably been a simple volvulus. But death almost always occurs in cases of internal strangulation if unrelieved. Manipulation, and inflating the bowel by means of bellows, have been suggested, but there is considerable risk attending these procedures. In *impacted gall-stone*, the progress is so rapid towards a fatal issue that operation, if undertaken, must be done immediately. The same remark applies to other foreign substances in the intestine.

The patient complains of CONSTIPATION *progressively increasing,* ABDO-MINAL PAIN, *and from time to time* VOMITING ; *there is general ill-health.* The case is one of CHRONIC INTESTINAL OBSTRUCTION.

§ 254. In Chronic Intestinal Obstruction (1) the abdominal pain is generalised, intermittent, and of increasing severity. (2) There is constipation, or a history of alternate constipation and diarrhœa culminating in complete stoppage ; and (3) abdominal distension in most cases, and peristalsis in some, may be visible. The chief causes of this condition are four in number :

I. MALIGNANT STRICTURE by new growth in the wall of the bowel—*e.g.*, cancer. Its most common situations are the colon, especially the sigmoid flexure, and the rectum. This cause of obstruction may be recognised by (1) the presence of a tumour or stricture which may be felt on examination *per rectum*, and the distension of the abdomen being most marked in the flanks. When the tumour is situated higher up than the sigmoid flexure, it may generally be felt through the abdominal wall ; and when situated in the sigmoid flexure, it may be inspected by a sigmoidoscope. (2) When the sigmoid flexure or rectum is affected, the illness is often preceded by sciatica on the left side. (3) There are cancerous cachexia, the age of the patient, and perhaps hæmorrhage, fœtid discharge and often ascites to aid in the diagnosis.

II. SIMPLE—*i.e.*, NON-MALIGNANT STRICTURE of the intestine may arise in consequence of dysenteric, syphilitic, or other ulceration, either in the colon or in the rectum. An ulcer alone is capable of producing

symptoms of obstruction. This cause is recognised by (1) the absence of a tumour, and (2) a previous history of dysentery (perhaps only a mild attack), and residence in a tropical climate ; or a history of syphilis, with a rectal discharge. Syphilitic stricture is rare, except between the sigmoid flexure and the anus.

III. PRESSURE ON THE BOWEL by a TUMOUR or an enlargement of some viscus such as the uterus. This cause is recognized by the physical signs of tumour or enlargement respectively.

IV. DILATATION OF THE BOWEL from paralysis of its coats. This is chiefly met with in the aged. It is differentiated from the other causes chiefly by (1) the absence of cachexia, tumour, emaciation, or other symptoms of the preceding causes, and an absence of a history of syphilis or dysentery. (2) The gradual formation of a soft faecal tumour, situated in the descending colon. The *Diagnosis* of these causes is also discussed in § 253.   •

V. CHRONIC PERITONITIS (§ 200) causes a matting together of the intestines, and intestinal obstruction may result. Cancerous peritonitis is attended by much pain and the effusion of much fluid ; but in tuberculous peritonitis there are mostly adhesions, less pain, and less fluid.

VI. CHRONIC INTUSSUSCEPTION is thus known : (1) It occurs usually in children ; (2) tenesmus is present ; (3) a tumour may be felt with characters similar to that met with in acute intussusception ; and (4) there is usually no marked distension (see also Acute Intussusception above).

VII. HIRSCHSPRUNG'S DISEASE (§ 252).

Prognosis.—In all forms of chronic intestinal obstruction the symptoms of acute obstruction are apt at any time to supervene, from impaction of faeces above the narrowing lumen of the gut, but apart from this the prospect differs considerably in the various causes. A cancerous stricture is the most, a dilated colon the least, serious. Syphilitic stricture may be relieved by iodides ; dysenteric stricture is irremediable. The course of a tumour varies with its nature. Chronic intussusception *may* spontaneously resolve, the invaginated part sloughing off and being passed by the rectum, but the outlook is always grave.

Treatment.—In most of the cases of chronic intestinal obstruction, surgical procedure is ultimately necessary, but at first the treatment consists in watching the patient until a diagnosis can be formed with as much accuracy as possible, and in giving digestible food, preferably such as leaves but little residue, and in relieving pain by opium and external applications (hot fomentations with turpentine or opium). In atony of the bowel, if oil enemata and other medicinal treatment fail, the faeces may require to be removed by mechanical means (scooped out). For simple stricture of the rectum gradual dilatation by bougies may be tried. In chronic intussusception operation is advisable. In cancerous stricture life may be prolonged by the formation of an artificial anus ; the longer the operation is delayed, the worse is the prognosis. It should never be delayed until vomiting has commenced. In some cases the bowel has been resected with success. The treatment of cancer is discussed in § 445.

CHAPTER XII

THE LIVER

WITHIN recent years much has been done with regard to the functional disorders of the liver, the structural diseases lend themselves very readily to physical examination and medical diagnosis The fact that the liver is capable of containing a fourth of the blood in the body is sufficient proof of its importance. All the blood passing from the stomach and intestines circulates through the liver, after which it joins the general circulation considerably altered in its composition. The liver aids in preparing proteins, carbohydrates and fats for the tissues of the body. The pancreas and the liver work in close co-operation; the pancreatic internal secretion passing direct to the liver. The important functions of the liver are the manufacture and the storage of glycogen, the secretion of bile, and a detoxicating action. Experimental research shows that the liver is also concerned in the manufacture of urea or its antecedents. Where the hepatic function is deranged the antecedents of urea are found in the urine—a high ammonia coefficient and increased amino-acids. The metamorphosis of the products of digestion in the course of their elaboration into urea is therefore one of the functions of the liver.

PART A SYMPTOMATOLOGY.

The symptoms due to disorders of the liver are not so clearly defined as those of cardiac or pulmonary diseases. The cardinal symptoms of structural disease of the liver are PAIN IN THE HEPATIC REGION, JAUNDICE, and a group of symptoms due to PORTAL OBSTRUCTION which include Ascites. When the liver cells become gradually destroyed, as in cirrhosis, serious disturbance of the general health ensues, and in the later stages of that and of some other hepatic disorders LETHARGY passing into coma supervenes. Functional derangement of the liver is always attended by DEPRESSION, which may amount to melancholia, and vague DIGESTIVE DISTURBANCES.

§ 255. Pain and Tenderness over the Liver are very marked in PERIHEPATITIS and any other condition in which the capsule is involved, and sometimes radiates upwards towards the right scapula. The onset of pain in the course of a liver complaint may therefore be of consider-

able importance ; for example, in hydatid of the liver, the natural course of which is painless, it would point to a danger of rupture of the cyst. When the upper surface of the liver is involved, the pain is very often *referred to the right shoulder ;* it is, indeed, a symptom of phrenic (diaphragmatic) irritation. The most severe form of pain, however, is that which occurs in connection with the passage of GALL-STONES (*biliary colic*). In a considerable number of hepatic disorders pain may be completely absent. There is, however, in many cases of marked disease or enlargement of the liver a feeling of weight or fulness in that region, accompanied by an inability to lie on the left side.

Hepatic pain may be *simulated* by Pleurodynia (rheumatism of the intercostal muscles), Intercostal Neuralgia, Pleurisy, Dyspepsia, and various gastric conditions, and by Intestinal or Renal Colic.

§ 255. Jaundice is the term applied to the yellow pigmentation of the skin and other tissues due to the non-elimination of bile. It appears first in the blood, then in the urine, in which bile pigments and acids may be detected (§ 308), next in the conjunctivæ, then in the skin universally and uniformly.

FALLACIES.—The yellow coloration of the conjunctivæ differentiates jaundice from all similar pigmentations of the skin. (1) Excess of *subconjunctival fat* may simulate jaundice, but this is readily distinguished by its unequal distribution. (2) The *sallowness* of the skin in chlorotic young women is easily distinguished by the absence of bile in the urine and yellowness of the conjunctivæ (3) The *cachexia* of carcinoma, malaria, and certain other forms of visceral disease, is differentiated in the same way. (4) The *bronzing* of the skin in Addison's disease is hardly likely to be mistaken for jaundice. (5) *Santonin* and *rhubarb*, administered internally, colour the urine, but do not give the reaction for bile in that fluid.

Symptoms accompanying Jaundice.—(1) Flatulent dyspepsia, and a bitter taste in the mouth. (2) Pruritus, which may be very troublesome in some cases ; eruptions, such as xanthelasma, are less common (3) The temperature, as a rule, is subnormal, and the pulse slow, (4) general debility and emaciation ensue in prolonged cases ; (5) mental depression is usual, and in severe cases, notably acute yellow atrophy and cirrhosis, cerebral symptoms such as delirium and coma, may appear towards the end, and xanthopsy (yellow vision) is sometimes present. (6) Hæmorrhages, either subcutaneous or from mucous membranes, are liable to occur in such cases.

Recent research has shown that jaundice can no longer be simply divided into obstructive and non-obstructive The causes of jaundice may be grouped under two broad types · (a) in which there is *extrahepatic obstruction*, and (b) in which there is *intra-hepatic* disease. Clinically, jaundice due to extra-hepatic obstruction is distinguished by the *colour of the stools*, which are pale, slate or clay-coloured, due to the absence of bile in the intestinal canal. There is also increased intestinal putrefaction and steatorrhœa, due to the increase of soaps and fatty acids in the stools.

(a) Jaundice due to Extra-hepatic Obstruction may be produced in

three ways—Obstruction within the bile-duct, disease in the wall, or pressure outside the bile-ducts.

I. FOREIGN BODIES within the duct, such as (1) gall-stones and inspissated bile; (2) hydatids, round worms, distoma, and other parasites; (3) foreign bodies from the bowel.

II. INFLAMMATION of the bile-ducts, usually spreading from the duodenum.

III. STRICTURE, or obliteration of the duct owing to (1) congenital absence; (2) perihepatitis; (3) cicatrisation after ulcer of the duodenum; (4) ulceration of the bile-duct, which may produce obstruction by the swelling around, or lead to stricture; and (5) spasmodic stricture (?).

IV. TUMOURS pressing on the duct, such as (1) cancer and other tumours of the liver; (2) enlargement of the glands in the transverse fissure of the liver; (3) tumours of the stomach, pancreas, kidney, great omentum; (4) fæcal masses in the intestines; (5) pregnant uterus; (6) ovarian tumours; and occasionally (7) tumours growing from the walls of the ducts.

(5) **Jaundice due to Intra-hepatic Disease** may originate from several factors. (i) Probably in the majority of cases, there is impairment of the hepatic function. The liver cells are so affected that they become inefficient and thus favour retention of bile pigment and salts in the blood. (ii.) Blood destruction (hæmolysis) leads to excess of bile pigment and the increased viscosity of the bile brings about temporary obstruction. (iii.) Catarrh and obstruction of the finer bile-ducts. The chief causes of intra-hepatic jaundice can be grouped under these headings: (1) cirrhosis of the liver; (2) bacterial poisons such as occur in catarrhal jaundice, pneumonia, syphilis, septicæmia, typhus, typhoid, relapsing fever, malaria, spirochætosis ictero-hæmorrhagica, yellow and other tropical fevers; (3) animal poisons such as ptomaines and snake-bite; (4) chemical poisons such as trinitrotoluol, tetrachlorethane (poisons affecting munition workers), phosphorus, arsenobenzol derivatives, nitrobenzene, chloroform and ether; (5) blood diseases with much hæmolysis, such as icterus neonatorum and acholuric jaundice.

Of these causes gall-stones and catarrhal jaundice are the most common. To *diagnose which cause* is in operation: 1. If possible, EXAMINE THE FÆCES, which are slate or clay-coloured in complete obstruction, and of normal colour in toxæmic jaundice. The presence of fat or parasites may assist in the diagnosis of the cause. But it must be remembered, as possible fallacies, that the fæces may become stained if mixed with urine; and that the bile-duct may be only partially obstructed, and enough bile may thus escape to colour the fæces.

2. Inquiry as to the HISTORY OF THE ATTACK. Jaundice coming on suddenly, especially in a middle-aged female patient previously in good health, almost invariably indicates obstruction by gall-stones (rare cases of nervous shock excepted). The intensity of the jaundice varies from week to week as the stones pass. Jaundice coming on slowly, and ulti-

mately becoming intense, is generally due to a tumour pressing on the hepatic duct, and is most often seen in association with cancer. A well-marked jaundice persisting some weeks is almost certainly obstructive. Occupation in a munition factory renders easy a diagnosis of the cause. A history of previous temporary attacks points in adult life to gall-stones ; in youth to " catarrhal jaundice."

3. EXAMINE THE HEPATIC REGION CAREFULLY. If the liver is enlarged cancer is the most probable cause ; interstitial hepatitis less commonly. If the gall-bladder is enlarged, cancer is more probable than stone. If ascites be present, the diagnosis rests between cancer and cirrhosis.

4. Inquire as to PAIN AND CONSTITUTIONAL SYMPTOMS. Pain of a spasmodic and severe character accompanies jaundice due to gall-stones and cancer. It is more constant and gnawing in character in congestion of the liver and catarrh of the bile-ducts. The temperature is not often elevated, but it may be so in catarrhal jaundice, jaundice due to poisons in the blood, pyæmic hepatitis, tuberculous affections, and local pus formations, such as inflamed hydatid. Cerebral symptoms are very rarely present, except when a fatal termination is at hand, unless the jaundice occurs in the course of pneumonia, fevers, or in that rare disease, acute yellow atrophy of the liver.

The *Prognosis* and *Treatment* of jaundice depend on its causal diseases (*q.v.*). The disappearance of bile from the urine indicates that the attack is coming to an end, though it may be some weeks before the skin clears. The flatulent dyspepsia and many of the concurrent symptoms may be relieved by the administration of ox gall (gr. 5 to 10 (0·3–0·6) with meals, together with carminatives. The itching of jaundice is often a most troublesome symptom, but it can generally be relieved by pilocarpine and alkaline lotions or baths.

By the time jaundice appears in a munition worker the condition is serious. Symptoms of acute toxæmia may develop, with delirium, coma and death. Prophylaxis consists in strict cleanliness of hands and food, abundance of milk and glucose and intermission of work in the trinitrotoluol department.

§ 257. Icterus Neonatorum is a mild transitory form of jaundice which affects a very large number (estimated by various observers at from 70 to 90 per cent.) of new-born infants. It appears usually on the second or third day of life, is not generally very intense, and rarely lasts longer than one or two weeks. The fæces are normal in colour, and apart from the jaundice the infant presents no other symptoms. The cause of the condition has been the subject of considerable debate, but the question is almost entirely an academic one, and the reader is referred to systematic works. No *treatment* is required.

Some cases of jaundice in the new-born have a much graver prognosis. (1) Kernicterus is the name given to the bile-staining of the nuclei at the base of the brain, which occurs in a particularly fatal form. This is familial in incidence. (2) Congenital syphilis, acting by stricture by the bile-ducts or otherwise or (3) congenital absence of the ducts. Both are fatal in a few months or less as a rule. (4) Winckel's disease and (5) Buhl's disease are rare forms of doubtful etiology. It should be remembered that jaundice in the new-born may be due to (6) sepsis. In stenosis of the ducts the stools are colourless ; with sepsis there will be other symptoms, but generally the differential diagnosis is not made during life

§ 258. Acholuric Jaundice (Cholæmia).—There may be no *symptoms*; it is a notable point in connection with the disease that the patients are often able to go about their work as if they were not the subjects of any abnormality. Symptoms when present are jaundice, weakness, a degree of anæmia, and splenomegaly, which may be extreme. These are liable to exacerbations in which the jaundice grows deeper, the anæmia and weakness more profound, and the general malaise may be associated with fever and perhaps vomiting. These attacks seem to be especially determined by cold. Hæmorrhages from the gums or stomach or into the retina are among the rarer symptoms of the disease. The blood changes are very characteristic: there are nucleated red cells even when the anæmia is very slight, great inequality in the sizes of the individual corpuscles, and predominance of cells showing basophil stippling. The blood cells are abnormally fragile—a point which clinches the diagnosis (§ 430). The blood also contains bile, whereas the urine contains only urobilin. The colour of the fæces is normal. The diagnosis from splenic anæmia may be very difficult (see § 439).

The *Etiology* is not known. The disease may be congenital or acquired. The former occurs in families, and may be transmitted by affected members of either sex.

The *Prognosis* of the congenital form is good as regards life, though recovery is not to be expected. In the acquired form the symptoms are more marked; the periods of health between the attacks are short, and the patient seldom lives to old age.

Treatment.—It is important to avoid cold and exposure. Splenectomy has now been proved successful in many cases and alone seems to hold out a prospect of permanent cure. The chief indications for it in the case of a patient hitherto at work are (*a*) increasing and disabling anæmia or debility, or (*b*) frequent or excessive pain.

PART B. PHYSICAL EXAMINATION

The liver lies chiefly in the right hypochondrium ; the left lobe extends across the epigastrium above the stomach into the left hypochondrium. The gall-bladder lies below, in contact with the liver, and is situated under the ninth right costal cartilage (see Figs. 68 and 71).

The routine methods of examination of the liver consist of INSPECTION, PALPATION, and PERCUSSION. Examination by X-rays may assist in the diagnosis of certain obscure tumours—*e.g.*, hydatid.

§ 259. Inspection locally teaches us but little, as a rule, unless the symmetry of the abdomen as observed from the foot of the bed be altered. However, the presence or the absence of *jaundice* should always be noted in cases of suspected hepatic disease. If slight, it may be noticeable only in the conjunctivæ and urine. Deficient chest expansion is noticed with inflammatory disease of the liver. The lower edge of the organ when enlarged may be seen moving with respiration. X-ray examination may reveal impaired movement, irregularities, and tumours of the organ. Note also if there are venous stigmata in the face or enlargement of the veins of the abdominal wall, such as occur with cirrhosis and portal obstruction.

During Palpation the patient should be placed in the recumbent posture, and, in order to obtain complete relaxation of the abdominal walls, he may be asked to " let his breath go." If this is not sufficient, the knees should be drawn up and the shoulders supported. Standing on the right side of the patient, place the palmar surface of the hand, previously warmed, on the right side of the abdomen, immediately above the iliac crest, pressing

it firmly yet gently inwards. The tips of the fingers should be inclined slightly upwards and inwards towards the median line, and the *upper margin of the index finger* should be pressed firmly down, working little by little upwards towards the costal margin. In this way the upper border of the index finger, always held perfectly flat, will come in contact with the margin of the organ if it be enlarged. But if it is not enlarged, the liver cannot be felt, for it lies altogether beneath the costal margin in the adult. In young children, however, the liver is proportionately larger in all its dimensions, and the lower edge normally protrudes beneath the costal margin. If the liver is enlarged, try to feel its surface by gently dipping the fingers down. Notice if its surface is smooth (as in fatty liver), or simply rough (" hobnail "). Umbilicated nodules may be felt in cancer of the liver. When there is fluid in the peritoneal cavity, this method of " dipping " the fingers (suddenly) is also useful ; but in most cases the finger tips only excite contraction of the abdominal muscles, and so frustrate our object. The other fallacies of hepatic enlargement are mentioned under *Percussion.* Note whether any tenderness or irregularity of the surface is present. Expansile pulsation of the whole liver is felt in cardiac disease with tricuspid regurgitation. The *gall-bladder,* if enlarged, may be discovered as a round elastic tumour, projecting beneath the ninth rib, at its junction with the cartilage at the edge of the right rectus muscle. Tenderness of the gall-bladder may be made out in this position when the patient sits up, leans forward and breathes deeply.

§ 260. **Percussion** should be light so as to elicit only the superficial or absolute dulness of the organ. In percussing the upper margin, start where there is a good lung note above, and percuss down from rib to rib in the nipple, mid-axillary, and scapular lines. Then repeat the process from space to space. In defining the lower edge, still lighter percussion should be used, and the examination should proceed from the tympanitic note of the intestine upwards towards the hepatic region. But the more certain method of detecting the lower edge is by palpation.

Normally, in the nipple line the *superficial* or absolute *hepatic dulness* commences two fingers' breadth below the nipple, and measures 3½ to 4 inches, and in a routine examination this is the most important measurement to obtain. The normal boundaries of the liver are given in Fig. 68.

The *lower border* arches upwards just beneath the right costal margin and crosses the epigastrium, where the hepatic dulness becomes continuous with the cardiac dulness. In the mid-sternal line the dulness extends from ¼ inch above the base of the xiphoid cartilage to about midway between the umbilicus and the xiphoid, where the lower edge may be felt by careful palpation when the abdominal wall is very lax. Thus the *absolute* dulness measures on an average about 2 *inches in the mid-sternal line* and 4 *inches in the nipple line.*

These landmarks do not indicate the deep dulness of the liver, which is more difficult, and in most cases less useful, to determine. But in some cases, such as abscess or hydatid, it is desirable to make out the deep (or *relative*) dulness of the

liver by heavy percussion. The extreme height of the liver, as thus made out, corresponds to the fifth rib in the nipple line, seventh space in the mid-axilla, and ninth space in the scapular line.

LIGHT PERCUSSION BOUNDARY (the one ordinarily used) gives the superficial or absolute dulness—*i.e.*, where the liver is in contact with the ribs.

	Nipple line	Mid-axillary line	Scapular line
Upper margin situated at the	6th	8th	10th rib.
Extent of dulness in vertical line	3½		3 inches

HEAVY PERCUSSION BOUNDARY gives the deep or relative dulness where the liver is covered by lung.

			Space
Upper margin situated at the	5th	7th	9th rib.
Extent of dulness in vertical line			4 inches.

FALLACIES.—The physician should never feel satisfied with mapping out the liver
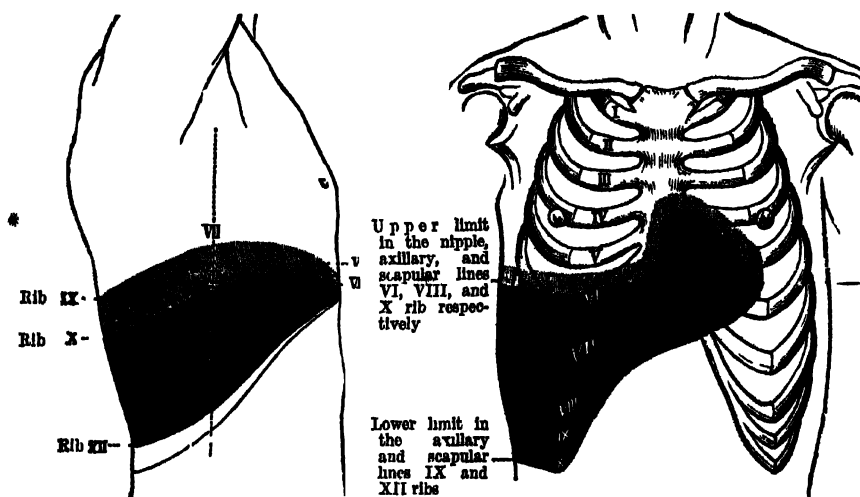
FIG. 68 —AREA OF LIVER DULNESS —The *superficial* (or absolute) dulness corresponds to the deep shading , the area of *deep* (or relative) dulness is larger and includes the lighter shading

once only, because the organ may be temporarily affected by many varying conditions, and the *percussion* boundaries by no means always give us a true index. Thus the lower edge may be masked by the dulness of the stomach after a full meal, by an accumulation of fæces in the colon, or by a thickened omentum. Great rigidity of the muscles, or œdema of the abdominal walls, may also obscure the lower edge of the liver.

The beginner, by *palpating* with his finger tips, which excite muscular contraction, generally fails to make out the lower margin of the liver, even when the organ is enlarged. By *percussing too heavily he fails to get the absolute dulness.*

Apparent *diminution* of the liver may arise from (i.) distension of the stomach or intestines with gas ; (ii.) by contractions of Glisson's capsule, especially on the under surface, giving rise to puckering or distortion of shape anteriorly ; or (iii.) emphysema of the lungs, which obscures the *upper* border very much. Great diminution or absolute loss of the liver dulness, owing to gas in the peritoneal cavity, is a diagnostic feature of perforation of the stomach or intestine.

Apparent *enlargement*, when attention is paid solely to the lower edge of the organ, may be due to a *displacement* of the liver downwards (i.) by pleuritic effusion, emphysema, or pneumothorax; (ii.) intrathoracic tumours; or (iii.) enlargement of the heart or hydro-pericardium. These and other fallacies may arise from paying attention solely to the *lower edge* of the organ; and, finally, the liver may in rare cases be dropped or "*floating*." "Riedel's lobe" is mentioned under Abdominal Tumours. Tumour or enlargement of the *gall-bladder* may be percussed as a dulness extending down from the liver to the umbilicus.

§ 261. **Fluid in the Peritoneum** (Ascites) is a frequent accompaniment of some hepatic disorders, and its presence or its absence must always be carefully noted. The methods of investigating this important matter have already been given (§ 208).

Ascites (Dropsy of the Peritoneum) is one of the **Evidences of Portal Obstruction**, and these are more frequently associated with some disease of the liver than of any other organ. Sometimes they are the only evidences we have of hepatic disorder.

The SIGNS OF PORTAL OBSTRUCTION are, in the order in which they appear : (1) A liability to attacks of *gastric* and *intestinal catarrh*, as evidenced by irritable dyspepsia, and the vomiting of mucus, streaked perhaps with blood, in the early morning before breakfast. (2) *Hæmorrhage*, sometimes in very large quantity, from the stomach and the bowels. (3) *Hæmorrhoids* may occur in other diseases (§ 250), but they are frequently associated with portal obstruction. (4) Attacks of *congestion of the liver*. (5) Congestion, and therefore enlargement, of the *spleen*. (6) *Ascites* (see below). (7) *Enlargement of the veins* of the abdominal wall from the establishment of a collateral circulation. (8) *Œdema* of the legs is a secondary and indirect result of the pressure of the ascitic fluid on the large veins within the abdominal cavity. (9) *Albumen* in the urine may arise in the same way, or from concurrent disease of the kidney.

Ascites, it will be observed, is a late sign of portal obstruction. It has already been fully described (§ 209), and it will be remembered that its three *principal causes* were Cardiac, Hepatic, and Renal disease. (1) In *cardiac disease* the ascites will have been preceded by dropsy of the legs ; (2) in *hepatic disease* the ascites is the predominating feature, though it may be followed by dropsy of the legs ; while (3) in *renal disorders* the ascites is only part of a dropsy which is general from the outset. *Cancer of the peritoneum* may also produce ascites, but here the nodules of cancer will probably be felt on palpation, and there will be other symptoms of cancer. Ascites may have to be diagnosed from an ovarian or other large abdominal cyst, and from fat in the omentum (§ 206).

Portal obstruction, and consequently ascites, are not present with equal frequency in all diseases of the liver, and in some they are absent. In order of frequency the causes are as follows :

(1) CIRRHOSIS, or an increase of the interstitial tissue (usually due to alcohol), is by far the commonest cause of portal obstruction, by producing pressure upon the minute branches of the portal vein within the liver.

(2) In CANCER of the liver portal obstruction and ascites are fairly

frequent, but they are due not so much to the cancer within the liver as to the pressure of enlarged glands in the transverse fissure of the liver upon the portal vein, or to secondary involvement of the peritoneum.

(3) PERIHEPATITIS may occasionally produce constriction of the portal vein by puckering at the fissure.

(4) SIMPLE CONGESTION of the liver may be attended by hæmorrhoids and gastric catarrh, but rarely by much ascites.

(5) Fatty and Waxy Liver, Abscess, and Hydatid are hardly ever attended by portal obstruction.

The other *Causes*, the *Prognosis*, and *Treatment* of Portal Obstruction have been described under Ascites (§ 209).

§ 262. Hepatic Efficiency Tests.—In cases of toxæmia it may be important to know whether the liver is functioning normally. Recent criticism tends to show that Widal's *hæmoclasic crisis test* is too sensitive for most practical purposes; the test is positive in cases other than purely liver disease. It is simple in application and depends upon the fact that when the liver cells are diseased the normal digestion leucocytosis is replaced by leucopenia. Count the white blood cells in the morning, give 7 ounces of milk on a fasting stomach; twenty minutes later, count again.

The most useful method of estimating inefficiency of the liver cells is the *lævulose tolerance* test. It depends upon the fact that when there is impairment of the efficiency of the liver, lævulose is not stored in the liver but passes into the systemic circulation, causing a rise in the blood sugar concentration. The blood sugar rises in proportion to the degree of hepatic inefficiency. Diabetes mellitus must be excluded before drawing conclusions from this test. The test is of value when it is necessary to decide whether jaundice is due to external obstruction or to disease of the cells of the liver. In purely obstructive jaundice there is little or no increase of blood sugar. Even when no clinical evidence is present in acute disease, this test may reveal liver inefficiency. The test aids when it is necessary to know whether the liver is affected in cases of amœbiasis, Addison's anæmia, cancer, obscure causes of jaundice and slight inefficiency is usually detected after a dose of arseno-benzol. According to the patient's weight, 30–50 gm. lævulose are given in water four hours after a light breakfast. The blood sugar is examined before the test, and at half, one and two hour intervals later, as in the glucose tolerance test. *Van den Bergh Test.*—Bile is not found in the urine when present in only small quantity in the blood. The bile may remain attached to the protein of the blood plasma, and therefore not be excreted by the kidneys. In such cases, the bilirubin of bile pigment may be detected in the blood by the Van den Bergh test, which consequently possesses diagnostic value. In cases of obstructive jaundice the so-called *direct reaction* to the test occurs; presumably the bilirubin has passed in a changed state from the efficient liver cells, and then been absorbed into the blood, owing to the obstruction below. When the bilirubin is returned

to the blood in an unchanged form, owing to inefficiency of the liver cells, the Van den Bergh test shows an indirect reaction. This, the indirect reaction, is met with in cases of toxæmia and in hæmolytic anæmias. Sometimes hepatic inefficiency and obstruction are both present; in such cases a double colour (biphasic) reaction occurs. When the bile is closely associated with protein it reacts little in the direct Van den Bergh test; when alcohol is used, this association is destroyed and the freed pigment gives the reaction.

For the test two solutions are required. (1) Concentrated HCl 15 c.c., sulphanilic acid 1 c.c., distilled water 1000 c.c. (2) Sod. nitrite 0·5 gm., distilled water, 100 c.c. Mix 25 c.c. of (1) to 0·75 c.c. of (2). For the *direct reaction*, 1 c.c. serum from the clotted blood is mixed with 1 c.c. reagent. A blue violet colour begins at once and attains its maximum in 10 to 30 seconds.

The *Indirect Reaction*. Add 2 c.c. of 96 per cent. alcohol to 1 c.c. serum and centrifugalise. To 1 c.c. of the clear fluid obtained, add 0·5 c.c. alcohol and 9·25 c.c. reagent. A violet red hue occurs, rapidly attaining its maximum. (Cp. *British Medical Journal*, 1922, I., pp. 716, 783.)

In all cases the urine should be examined for bile, and sometimes for urea, leucin and tyrosin.

In obscure cases it may be necessary to estimate the percentage of cholesterol in the blood. Another means of investigation of the liver is carried out by Einhorn's tube. The tube is passed into the duodenum. A concentrated solution of magnesium sulphate is taken by mouth and causes a rapid flow of bile. The duodenal contents can be withdrawn and examined for pus, etc.

X-ray examination is very useful in cases of tumour and gallstones, and also reveals enlargement or diminution of the liver.

PART C. DISEASES OF THE LIVER

Routine Procedure.—FIRST : Ascertain *what is the patient's Leading Symptom*. The symptoms of disorder of the liver we discussed in Part A. —*e.g.*, gastric disturbance, pain (or a feeling of weight or discomfort in the hepatic region), or jaundice. If there be severe and paroxysmal pain, turn first to biliary colic (§ 266).

SECONDLY : Learn the *History* of the patient's illness, eliciting the facts in chronological order; and in this way ascertain the important fact whether the disease be *acute* or *chronic*, because disorders of the liver may be conveniently classified into these two groups.

THIRDLY : THE EXAMINATION OF THE LIVER must next be made. The routine method is as follows :

Ascertain : 1. Whether the liver is *enlarged*, locally or generally, or *diminished* (by percussion in the nipple line, and abdominal palpation), and whether there is any *pain, tenderness*, or other abnormality ; 2. Whether there be any *fluid* in the peritoneum (§ 208) ; 3. If there is any *jaundice* (§ 256) ; 4. Examine *the urine* for bile pigments, urates, etc. 5. In certain cases the hepatic efficiency tests and X-ray examination must be carried out.

Classification.—For clinical purposes, diseases of the liver may be conveniently divided into Acute and Chronic Disorders.

If the illness is one of long standing, and has come on insidiously, the reader should turn to Chronic Diseases of the Liver (§ 273). The acute will be first described.

ACUTE DISEASES OF THE LIVER

If the illness has come on more or less suddenly, and is attended by considerable malaise or other constitutional symptoms, it is one of the acute diseases of the liver or bile ducts, probably: I. Acute Congestion. II. Catarrhal Jaundice ; III. Spirochætosis Ictero-hæmorrhagica ; or IV. Gall-stones. The less common acute diseases are : V. Perihepatitis ; VI. Abscess ; and VII. Acute Yellow Atrophy.

I. *The patient complains of* pain *or* discomfort in the hepatic region, *the liver area may be* increased, *slight jaundice and numerous vague* dyspeptic symptoms *are present, but there is little or no fever.* The disease is probably Acute Congestion of the Liver.

§ 263. Acute Congestion of the Liver.—Clinically, there are two kinds of congestion of the liver—an active or acute congestion, and a passive or mechanical congestion. *Active* or *arterial* congestion is usually met with in the form of acute attacks due to dietetic errors ; though it may sometimes occur as a more subtle and sometimes latent condition in a subacute or chronic form which eventuates in cirrhosis. *Passive* or *venous* congestion is due to obstructed venous return (mostly in chronic cardiac or pulmonary disease) ; it is in the nature of things a chronic process, and is considered under Chronic Diseases (§ 255).

Symptoms.—(1) The onset is usually somewhat sudden, after a series of indiscretions in diet, especially in the matter of alcohol. The patient complains of pain, or a feeling of weight or uneasiness in the region of the liver, and he may be unable to lie on the left side. (2) There is generally a slight but uniform enlargement of the liver, and some degree of tenderness. (3) Slight jaundice is present on the second or third day in the majority of cases, but it is never so intense as in catarrhal jaundice or gall-stones. The fæces are dark in colour, owing to the presence of bile. (4) Certain gastro-intestinal symptoms are present—nausea, headache, furred tongue, a bitter taste in the mouth, and flatulence ; the bowels are usually constipated ; the urine is scanty, high coloured, and deposits urates on standing ; and, there is usually some depression and irritability of temper.

Etiology.—(1) By far the most frequent cause is alcoholic excess. Constant indulgence in rich foods containing fat, sugars, and spices may also produce congestion. (2) Residence in hot climates, especially when associated with malaria and dietetic errors ; but many attribute to the climate what is really due to alcohol or faulty diet. (3) Suppression of an habitual discharge, especially bleeding piles or menstruation ; and

TABLE XVIII.—ACUTE DISEASES OF THE LIVER.

	Jaundice.	Enlargement of the Liver.	Ascites.
I. ACUTE CONGESTION . .	Not very great.	Slight increase.	Usually absent.
II. CATARRHAL JAUNDICE .	Always present and marked.	Slight increase.	Absent.
III. SPIROCHÆTOSIS ICTERO-HÆMORRHAGICA.	Usually marked, occasionally absent	Moderate.	Absent.
IV GALL-STONES	Very marked in most cases.	May be considerable increase	Absent.
V. PERIHEPATITIS . . .	Absent.	None unless another cause	Usually absent.
VI ABSCESS OF LIVER . .	Generally present.	Moderate and irregular enlargement	Usually none.
VII. ACUTE YELLOW ATROPHY.	Very marked.	Liver diminished in size.	Absent.

(4) dysentery and febrile states are often accompanied by congestion. (5) Sudden or protracted chill and (6) injury have been mentioned as causes. An attack of acute congestion may be *predisposed to* by (i.) the presence of chronic congestion (§ 280) ; (ii.) previous attacks of malaria ; (iii.) indolent or sedentary habits.

The *Diagnosis* is based upon the occurrence of symptoms of gastro-intestinal disturbance in association with pain and enlargement of the liver. In *perihepatitis* the first-named are absent, the pain is much more acute, and syphilis is probably in operation. The diagnosis from the other acute hepatic disorders is given in Table XVIII above. The symptoms of *pleuro-pneumonia* at the onset may include jaundice and the other symptoms of acute congestion of the liver, for which, indeed, this disease may be mistaken. It is important, therefore, to examine the base of the right lung in all such cases when associated with jaundice.

Prognosis.—Acute congestion is very apt to recur, especially if the patient continues his dietetic indiscretions. The intervals between the attacks become shorter, and the condition is followed by chronic congestion, and, eventually, cirrhosis. An attack of moderate severity rarely lasts more than a week or two. Unless a condition of cirrhosis is reached (when enlargement is checked to some extent by the shrinking of the newly-formed fibrous tissue), the degree of congestion may be fairly estimated by the amount of enlargement.

Treatment.—The indications are (1) to relieve the congestion of the portal system, and (2) to correct dietetic errors. To relieve the congestion, saline purgatives are specially indicated, such as the sulphates of magnesia, potash, soda, or the bitartrate of potash. Carlsbad, Friedrichshall, or Hunyadi Janos water should be taken every morning early, and a full dose of calomel, podophyllin, or pil. hydrarg. at night. In severe cases leeches, or dry or wet cupping in the region of the liver, may relieve the

pain considerably. Leeches are sometimes applied to the margin of the anus, but this is not always convenient. Murchison recommended ammonium chloride, gr. xx. (1·3) t.i.d., to induce free diaphoresis, and diminish the portal congestion and pain (F. 46, 51, 53, and 66 may be useful). Ipecacuanha is in great repute among Indian physicians, and is given in large doses, as in dysentery. For the gastric symptoms, alkalies, carbonate of magnesia and bismuth are useful. Nitrohydrochloric acid and nux vomica are useful in convalescence. The diet during the attack should be of the simplest, consisting at first of 2 pints of milk a day. Alcohol in any form should be strictly forbidden.

II. *The patient, who is* YOUNG, *has suffered from* GASTRO-INTESTINAL DISTURBANCE *for some days or weeks, when* JAUNDICE, WITH CLAY-COLOURED STOOLS, *sets in somewhat suddenly, without local pain, and with little or no enlargement of the liver.* The disease is probably CATARRHAL JAUNDICE.

§ 264. **Catarrhal Jaundice,** though called Acute Cholangitis, is jaundice due probably to interference with the function of the liver cells.

Symptoms.—(1) The jaundice is usually of sudden onset, though it is preceded for a shorter or longer time by signs of gastro-intestinal disorder. (2) The jaundice is often very intense, but in mild cases may be slight. It generally begins to subside in the course of two or three weeks. If it lasts longer, and especially if accompanied by fever, some other cause (§ 231) should be suspected. (3) The stools are pipe-clay coloured, and the urine is dark with bile. (4) Nausea and loss of appetite, flatulence, and constipation are generally present. (5) A feeling of uneasiness or weight in the hepatic region is usually complained of. There may be slight enlargement of the liver, the edge being smooth, firm, and tender. The spleen may be slightly enlarged. (6) There may be slight fever at the commencement, but it usually subsides before the patient is seen, and the pulse is abnormally slow.

Etiology.—(1) Until the lævulose test proved that the hepatic function was deranged, this disease was supposed to be due to catarrh ascending from the duodenum to the bile-ducts. (2) It is by far the commonest form of jaundice met with in children and young adults. (3) Exposure to chill. (4) Secondary to congestion (§ 263) or cancer. (5) It frequently follows the passage of a gall-stone. (6) In the adult gout or gouty conditions predispose. (7) Epidemic catarrhal jaundice is a closely allied variety, of unknown cause, which occurs in epidemic form. It assumes large proportions only when men are massed together, as in armies. Some of these cases, however, may be in reality " Weil's disease " (§ 265).

Diagnosis.—Catarrhal jaundice may have to be diagnosed in an old person from *cancer*, but in the latter the jaundice comes on slowly, with pain, and it lasts many months. In *gall-stones* there is biliary "colic " (§ 266). In *congestion* of the liver the jaundice is less marked, and the fæces are not clay-coloured.

Prognosis.—The disease is never fatal. It usually terminates in a few

weeks.   The outlook is unfavourable only when catarrhal jaundice complicates other maladies.

Treatment.—Remove any cause of the concurrent gastro-enteritis, and allay the condition with alkalies, alkaline carbonates, rhubarb, or bismuth. A brisk mercurial purge, followed by a saline twice a week, relieves the congestion of the intestines and the liver.   Ox-gall, creosote, or salol are sometimes useful as antiseptics.   Sodium salicylate with ammonium chloride, āā gr. x. (0·65), is very effective.   Rectal injections of one or two pints of water daily (60° to 90° F.), retained as long as possible, allay intestinal irritation.   The treatment for Congestion (§ 263) is applicable.

III. § 265. **Spirochætosis Ictero-Hæmorrhagica** (synonyms : Acute Infective Jaundice, Weil's Disease, Epidemic Jaundice, Mediterranean yellow fever).   During the European war epidemics of jaundice occurred in the various theatres of hostilities.

Symptoms.—The malady comes on suddenly, with marked headache and prostration.   The muscular pains in back, chest, and legs are very severe.   Jaundice appears about the third to the fifth day, moderate in degree or intense, lasts about ten days and gradually disappears.   The stools are clay-coloured.   The liver and spleen are enlarged ; the urine contains albumen, epithelial casts, and sometimes blood.   The temperature reaches 103° or 104° on the second or third day, begins to fall about the fifth day, and becomes normal about four to six days later.   After an interval of three to eight days there may be a recrudescence of fever, but the relapse is less severe than the initial fever.   In some cases there are complications, such as urticarial, erythematous and purpuric rashes, epistaxis and other hæmorrhages.

Etiology and Varieties.—Weil described a form of acute febrile jaundice in 1866 which was rarely met with in this country.   Hyperpyrexia, severe hæmorrhages, and uræmia were sometimes associated with it.   Recently, Japanese observers described a form of febrile jaundice clinically identical with Weil's disease, occurring endemically and epidemically, and proved to be due to a spirochæte.   Hence it was named Spirochætosis Ictero-hæmorrhagica.   The same organism was detected in some of the cases of febrile jaundice occurring on the Western front.   The spirochæte is found in the blood only in the early days of the disease ; in the fatal cases it is found in the liver and kidneys.   The urine contains the spirochæte—an important point to remember in prophylactic measures.   It is suspected that the spirochæte enters the body by insect bites, abrasions or drinking water.

Diagnosis.—It should be remembered that all cases with acute febrile jaundice are not necessarily Spirochætosis Ictero-Hæmorrhagica.   Jaundice with fever occurs with simple catarrhal jaundice due to chill, and it may occur in association with other acute fevers, especially typhoid, paratyphoid, malaria, or relapsing fever.

The *prognosis* depends upon how much the kidneys are involved.

Treatment is as for catarrhal jaundice.

IV. *The patient, usually an elderly female, is suddenly seized with* PAROXYSMS OF SEVERE PAIN *in the hepatic region, and in the course of twelve to twenty-four hours she becomes* JAUNDICED, *the stools becoming clay-coloured.*   The attack is one of BILIARY COLIC.

§ 266. **Gall-stones** and **Biliary Colic.**—Gall-stones are concretions which form in some part of the biliary passages, most commonly in the gall-bladder.   CHOLELITHIASIS is the condition in which gall-stones are developed.   When gall-stones move along any of the ducts, they give rise to Biliary Colic.

GALL-STONES may vary in size from a sand-grain to a golf-ball.   When solitary,

they are round or oval in contour. The facets or flattenings of their surface are caused by the pressure of one against the other; this indicates that there has been more than one stone in the gall-bladder or bile-ducts. The colour varies from yellow to dark brown; their chief physical characteristics are the smooth " soapy " surface, the ready way in which they crumble between the thumb and finger (though sometimes they are very hard), and their lightness as compared with renal calculi. They generally consist chiefly of cholesterin mixed with calcium and bile pigment, but are sometimes pure cholesterin, pure bilirubin, or pure calcium carbonate. Cholesterol is contained and held in solution by bile salts in normal bile. When from obscure causes the liver is unable to produce the bile salts in sufficient quantity, there is a high cholesterol content in the blood and bile, with eventual deposition of cholesterol and formation of gall-stones. Normal individuals can eat food containing cholesterol, because more bile salts are produced by the liver and hold the cholesterol in solution. With other individuals this capacity is defective. The foods which increase the cholesterol content of the blood are : egg yolk, butter, cream, liver, kidney, pancreas, brain and fats.
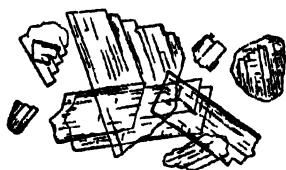
FIG. 69 — Cholesterin Crystals. Microscopic appearance presented by fragments of gall-stones in the fæces or in the duodenal tube.
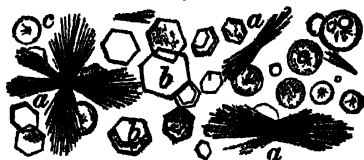
FIG. 70 — a, TYROSIN, in bundles of needle-shaped crystals, and c, LEUCIN, spherical crystals with concentric markings, found in the urine in rare cases of acute yellow atrophy of the liver. b, CYSTIN (clear six-sided plates) is a rare urinary deposit due to an inborn error of metabolism. It may form renal calculi.

Biliary Colic.—Symptoms may be absent when the stone is at rest, but when it begins to move (i.) the pain is agonising ; it starts in the epigastrium and shoots into the right hypochondriac region towards the spine and up to the right shoulder, but never passes downwards. The paroxysm is usually so severe that the patient is in a state of partial collapse, with vomiting, hiccough, subnormal temperature, and a quick, weak pulse. Sometimes there is a rigor, and the temperature rises a few degrees. Between the paroxysms of acute pain there is a constant dull aching and tenderness over the hepatic region. The attack lasts from a few hours to a few days. (ii.) The liver may be enlarged and if a stone becomes impacted in the hepatic duct the enlargement may be considerable. (iii.) Jaundice usually appears twelve to twenty-four hours after· the paroxysm, and lasts from a few days to a few weeks. It is most intense when the stone is impacted in the common duct.

The *Symptoms* which arise vary somewhat with the *position of the gall-stone* (Fig. 71). Thus : (i.) If a stone is impacted in the *common duct*, there are biliary colic, marked jaundice, and a distended gall-bladder, and if the impaction continues the liver becomes enlarged. (ii.) If a gall-stone be impacted in the neck of the gall-bladder (*i.e., in the cystic duct*), biliary colic is present without jaundice. In time the gall-bladder may be dis-

tended with mucus, and form a definite abdominal tumour. (iii.) Stone impacted in the *hepatic duct* is rare. It causes biliary colic and jaundice, but the gall-bladder is not distended. (iv.) Stones occasionally form in the radicles of the hepatic ducts, and give rise to indefinite symptoms, sometimes without pain, and usually without jaundice. (v.) Sometimes small particles of cholesterin (biliary sand) in the *gall-bladder* may give rise to recurring paroxysms of pain, unaccompanied by any other symptoms which defy diagnosis.

Diagnosis of Biliary Colic.—It is distinguished from the two other forms of colic in Table XIII.—§ 196. The severity of the pain and its paroxysmal character usually distinguish it from other acute diseases of the liver. Pseudo-biliary colic is sometimes met with in nervous women. The diagnosis from *cancer* of the liver may be very difficult. Both occur at the
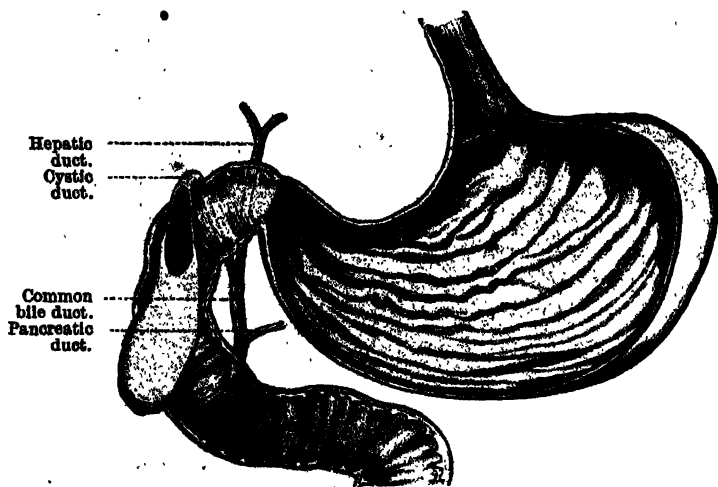
FIG. 71.—The STOMACH AND DUODENUM opened to show the ducts in connection with the Liver and Pancreas.

same age, and both cause jaundice ; further, cancer may follow after years of trouble from gall-stones. In cancer, jaundice steadily becomes more intense. It must be remembered that in some cases gall-stones are passed without colic, but with jaundice ; consequently, *recurring attacks* of jaundice in an elderly woman should lead one to suspect gall-stones. In expert hands a radiogram may show gall-stones, but a negative plate is not conclusive. In all suspected cases the stools should be carefully examined for stones. *The presence of ascites* points to cancer, which rarely exists long without peritoneal effusion.

The *symptoms* of gall-stones AT REST IN THE GALL-BLADDER are very obscure, and as they occur often in elderly women are frequently mistaken for cancer. (1) The most frequent symptom is dyspepsia, continuous or intermittent. The patient has discomfort and pain at varying intervals ;

some days immediately after food, on other days, one to several hours
after a meal. It is not relieved by alkalies, but often by a glass of hot
water. Nausea, vomiting, headaches and cold sweats may occur. (2)
Attacks of abortive biliary colic may occur from time to time after fatigue
or exertion. (3) Attacks of "biliary fever," i.e., shivering, with slight
temperature, may occur at intervals for months or years. (4) The pain
and discomfort is usually in the epigastrium, and there may be tenderness
at the angle of the right scapula. Between attacks of colic, tenderness
and aching are felt in the areas supplied by the seventh to the ninth dorsal
spinal segments, the areas which supply the sympathetic nerves to the
gall-bladder and bile ducts. (5) For the rest, the symptoms are negative,
no jaundice, ascites or other symptoms—merely a condition of general
ill health caused by septic absorption from the gall-bladder. (6) Hyper-
chlorhydria often accompanies disease of the gall-bladder and bile ducts ;
frequent regurgitation of bile into the stomach indicates chronic disease
of the gall-bladder. (7) The stones may become encysted, but more
often, without surgical intervention, ulceration, perforation, abscess and
fistula result. Cancer of the gall-bladder is usually preceded by gall-
stones.

Etiology.—(i.) Gall-stones occur usually after the age of 50 ; (ii.) are
more common in women and (iii.) in stout persons of sedentary habit
whose diet is rich in fat and sugar. (iv.) There is often a history of gout,
asthma or migraine. (v.) They may follow cholecystitis due to typhoid,
coli or streptococcal infection, or any cause of stagnation of bile in the
gall-bladder, as occurs in visceroptosis. (vi.) An attack of biliary colic
is often determined by a sudden strain or overloaded stomach.

Course and Prognosis.— The prognosis as to recovery from an attack of
biliary colic is excellent, but recurrence may be expected. A stone usually
forms in the gall-bladder and becomes impacted for a time in the neck of
the cystic duct, giving rise to biliary colic without jaundice. It then
passes down the common duct, and hence causes jaundice. This rarely
lasts more than a few weeks, but rare cases have been reported where it
lasted two years. Impaction for any length of time leads to consequences,
which may be classified thus : (i.) *Ulceration* of the ducts, with pyrexia, or
abscesses of the liver and bile-ducts, and consequent subacute pyæmia ;
(ii.) *perforation* into adjacent tissues, leading, for example, to fatal peri-
tonitis ; (iii.) inflammation and *abscess* of the gall-bladder, which may open
externally, perforate into the peritoneum, or ulcerate into the intestines ;
(iv.) formation of *fistula* between the gall-bladder and the colon or duo-
denum, through which stones can pass of such a size that they may cause
intestinal obstruction.[1] Cancer may supervene in later years.

Treatment.—During the attack anodyne treatment is called for. . Opium

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[1] Large gall-stones may gradually ulcerate through from the gall-bladder to the
duodenum, in some cases almost without symptoms. Murchison collected some thirty-
four of such cases where the gall-stones were large enough to give rise subsequently to
intestinal obstruction.

or a hypodermic of morphia and atropine should be given (F. 25). Chloro-form inhalations are used in severe cases. Other drugs recommended are chloral, spt. ætheris, and ext. belladonnæ, gr. ⅙ (0·03), every two hours. 15 drops of adrenalin 1 in 1000 relaxes muscular spasm and hence allays pain. Where there is hyperchlorhydria, a tablespoonful of olive oil with a small dose of tincture of belladonna is useful, given before meals. Hot water with ℥i (4 gm.) of bicarbonate of soda to the pint aids the flow of bile, and hot turpentine stupes relieve pain. Sometimes an attack of pain is warded off by putting the patient into a hot bath (100° F.).

Between the attacks the diet must be supervised. Foods containing cholesterol must be omitted, therefore most fats are forbidden. A little butter is allowed, but no cream or yolk of egg, nor kidney, liver, brain, sweetbread or the fat of meat, pork, goose and duck. A prolonged course of alkalies and sodium salicylate does good. A sudden rush of bile can be obtained, in order to clear the biliary passages, by giving magnesium sulphate in doses of ℥i to ℥iv (4 to 16) in concentrated solution before breakfast. Urotropin and sodium salicylate are excreted in the bile ; the latter increases the excretion of bile salts and cholesterol ; the urotropin sterilises the infected bile when given in large doses, with citrate of potassium which prevents bladder irritation. Surgical treatment is indicated where there is suppuration, when the gall-bladder remains dis-tended, the common duct is blocked, or biliary colic frequently recurs.

§ 267. Diseases of the Gall-bladder are chiefly manifested by pain and enlargement or swelling of the gall-bladder, which first appears just beneath the tip of the ninth right costal cartilage. For the fallacy of Riedel's lobe see Abdominal Tumours, § 211.

A. Catarrhal Inflammations : (a AOUTE CATARRH (which corresponds to Catarrhal Jaundice, § 264) ; (b) CHRONIC CATARRH. B. Suppurative Inflammations : (a) SUP-PURATIVE CATARRH, which may consist of—(a) Simple empyema, and (β) suppurative cholangitis ; (b) ULCERATION, PERFORATION, FISTULA, and STRICTURE of the gall-bladder and bile-ducts ; (c) ACUTE PHLEGMONOUS INFLAMMATION and gangrene of the gall-bladder.

CHRONIC CATARRH of the gall-bladder presents symptoms resembling those due to gall-stones within it (vide supra), but there is less pain, very slight jaundice, and no tenderness on pressure over the region of the gall-bladder.

SIMPLE EMPYEMA of the gall-bladder, without involvement of the hepatic ducts, is nearly always due to gall-stones. There is swelling, with continual localised pain and tenderness ; and the abscess may burst in various directions, or point externally.

SUPPURATIVE CHOLANGITIS is practically indistinguishable from pyæmic abscesses (§ 269). There is enlargement of the liver, and tenderness.

ULCERATION of the gall-bladder is referred to above under Gall-stones and PER-FORATION is usually a result of the same lesion.

STRICTURE is generally also a consequence of the ulceration following gall-stones, but it may sometimes be due to other lesions (see § 256). The result depends upon the position of the stricture. In the cystic duct it leads to distension of the gall-bladder. In the common duct it leads both to distension of the gall-bladder and considerable enlargement of the liver. It is rare in the hepatic duct, where it produces enlargement of the liver.

ACUTE PHLEGMONOUS INFLAMMATION of the gall-bladder is a rare affection. It comes on suddenly, with symptoms resembling perforative peritonitis, and is difficult to diagnose from acute appendicitis. Sloughing of the wall of the gall-bladder may

occur with consequent cholangitis and local or general peritonitis. It is usually rapidly fatal unless dealt with surgically.

MEMBRANOUS or FIBROUS CHOLECYSTITIS, secondary to retained gall-stones, has been recorded by Sir Humphrey Rolleston; a cast of the gall-bladder is passed with hepatic colic. The condition is diagnosed from gall-stone by finding the cast in the stools.

The less common **Acute Disorders** of the Liver remain to be considered, viz., PERIHEPATITIS, ABSCESS OF THE LIVER, and ACUTE YELLOW ATROPHY.

IV. *The patient complains of* PAIN AND TENDERNESS *in the hepatic region, aggravated by movement. There is* NO JAUNDICE, *and other hepatic symptoms are absent.* The malady is probably PERIHEPATITIS.

§ 268. **Perihepatitis** is inflammation of the capsule of the liver, which becomes opaque and thickened, and by its contraction may lead to considerable distortion of the shape of the liver.

Symptoms.—(i.) Acute attacks usually set in suddenly, with pain in the hepatic region, radiating to the shoulder, and there is tenderness, increased on movement, pressure, or cough. (ii.) Fever is absent as a rule, and the patient may appear to be in his usual health. (iii.) Friction may be felt or heard. (iv.) Unless some other disease is present, there is no jaundice. Recurrent attacks lead to thickening of the capsule, recurring ascites, necessitating repeated tapping, and occasionally jaundice. The puckered liver, with its thickened, rounded, distorted edge, can sometimes be made out. The history of a *Cause*, especially *syphilis*, is usually obtainable. It is sometimes part of an inflammation of the liver itself, or is associated with an abscess, tumour, or cirrhosis. Sometimes the inflammation extends from adjacent organs, as in pericarditis, pleurisy, or gastric ulcer, or it may be part of a general peritonitis. Perihepatitis occasionally complicates acute or subacute rheumatism.

Diagnosis.—The characteristic pain and the absence of jaundice differentiate it from many other liver diseases. The conjunction of syphilis is also very helpful. Cases of cysto-cholelithiasis (§ 266) or gumma of the liver may at times be mistaken for perihepatitis.

Prognosis.—Simple cases tend to recover. In cases which have lasted for a long time a certain amount of cirrhosis of the liver ensues. Portal obstruction may ultimately result from puckering at the fissure, and considerable distortion of the liver may result in the same way.

Treatment.—The diet must be spare, and the patient must be kept warm. Salines are given, with blue pill and rhubarb. Externally, hot fomentations and poultices give relief, and if the pain is severe, leeches are recommended. The cause when known must be treated—*e.g.*, syphilis with iodide of potassium.

V. *There is* ENLARGEMENT *of the liver, accompanied by* PAIN *and tenderness, and the boundaries of the area of dulness are* IRREGULAR ; *there are* SHIVERINGS, SWEATING, *and* INTERMITTENT PYREXIA. The disease is ABSCESS OF THE LIVER.

§ 269. **Abscess of the Liver.**—Solitary or multiple collections of pus may occur in the liver, due to septic infection, to suppuration of the bile channels, or portal vein, or more rarely to suppuration of pre-existing morbid conditions, such as hydatids or gummata. "Tropical" abscess occurs after amœbic dysentery, a common cause in the tropics ; it is usually solitary, whilst pyæmic abscesses are usually multiple.

Symptoms.—(i.) The onset is usually *acute*, with pain and tenderness of the liver, accompanied perhaps by a dry cough, with shallow respiration and digestive disturbance. The pain is affected by respiration, and is worst when the patient lies on the left side. (ii.) The liver is enlarged, and the enlargement may extend downwards, or more often upwards, even to the nipple. There may be fluctuation. (iii.) More or less jaundice is present as a rule. (iv.) Constitutional symptoms are marked. There

is usually high fever, continuous at first, then with increasing oscillations. Rigors and sweats are common. Later on the patient falls into the typhoid state, with emaciation, vomiting, diarrhœa, and delirium.

Besides the acute type just described, there is an *asthenic* variety, with insidious onset, general failure of the health, and periods of continuous or intermitting fever, followed by intervals of apyrexia, resembling malaria. Cough and dull aching over the liver and in the right shoulder are generally present from the beginning. The tropical abscess occasionally has no symptoms.

Diagnosis.—(i.) The pain and pyrexia distinguish abscess from *hydatid* (when not in a suppurating condition). (ii.) A distended and *inflamed gall-bladder* is recognised by a history of gall-stones, and its outline may be palpable on examination. (iii.) Abscess is often mistaken for severe *malaria*. But malaria is amenable to quinine, the elevations of temperature are periodic, and each paroxysm has three stages. (iv.) A hepatic abscess may be diagnosed from other swellings of the liver by exploratory aspiration, giving the reddish " anchovy sauce " distinctive pus. (v.) A low form of pneumonia at the base of the right lung so frequently accompanies liver abscess that its presence is an important aid to diagnosis in obscure cases. (vi.) X-ray shows upward enlargement of the liver when the right lobe is involved and limited movement of the diaphragm.

The insidious cases of liver abscess are always difficult to diagnose, and where health is deteriorating, with obscure pyrexial conditions from time to time, almost every general or local inflammatory disorder may be suspected before liver abscess. On the other hand, cases have occurred where medical men, diagnosing abscess, have explored the enlarged livers of *leucocythæmia* and *pernicious anæmia.* This mistake may be avoided by examining the blood before resorting to puncture. Slight increase of the polymorphonuclears indicates abscess.

Etiology.—Hepatic abscess, single or multiple, may arise from—(i.) Suppuration in a pre-existing focus of disease—*e.g.*, hydatid, gumma, tuberculous abscess, actinomycosis, or malignant growth ; (ii.) ulceration of the biliary passages such as occurs in cholecystitis ; (iii.) ulceration of the alimentary canal. In this case the abscesses are usually multiple, except in amœbic dysentery, when there is one large abscess, the contents of which are sterile except for the presence of the amœba. Such an abscess may become secondarily infected with staphylococci, etc. (iv.) Inflammation and pus-formation in the abdomen, especially in cases of old-standing suppuration of the pelvic organs and in appendicitis. (v.) Occasionally operations on the rectum or in any septic area produce an abscess in the liver, consequent on the conveyance of a septic embolus by the portal vein. (vi.) Pyæmia. (vii.) Trauma in a few cases.

Prognosis.—(1) The case mortality varies from 57 to 80 per cent. Death usually takes place in three weeks in cases with multiple abscesses. The pyrexia increases, and the patient dies in the typhoid state. (2) Solitary abscess may lead to death in a month, or the patient may live for one or two years, with obscure symptoms as described above. (i.) The abscess may burst into the peritoneum, pericardium, or alimentary canal, with a fatal issue, or it may open externally and gradually recover by free discharge. (ii.) Frequently the abscess, especially a " tropical " abscess, bursts into the right lung or the pleura. The patient develops a severe cough, with signs of consolidation of the right pulmonary base, and the abscess contents are brought up as a red-coloured sputum. Recovery may result, or the continued discharge may lead to death from exhaustion or lardaceous disease.

Treatment.—When the evidence points simply to acute inflammation of the liver, before the temperature leads one to suspect pus formation, anodyne treatment, such as cupping and hot poultices are employed. Ammon. chlor. gr. 20 (1·35) t.i.d. is the best drug. With a history of amœbic dysentery, give emetine gr. 1 (0·06) twice daily. Saline purgatives, spare diet, and absolute rest in bed are necessary. As soon as an abscess is suspected, exploratory puncture must be performed ; at least six punctures should be made before abandoning the attempt to find pus. If an abscess is discovered, free drainage must be established. Sir Leonard Rogers has replaced drainage by

flushing out two or three times daily. Aspiration may be performed and emetine (1 in 1000) injected into the abscess cavity.

§ 270. **Subphrenic Abscess** (Abscess beneath the Diaphragm).—The *Symptoms* are much the same as those of tropical liver abscess. When occurring above the right lobe, the liver dulness is continued up in the axilla, perhaps as far as the level of the nipple, and is convex, or dome-shaped, upwards. The base of the right lung shows signs of congestion, and there are evidences of pleurisy at one or both bases.[1]

Etiology.—In men the most common causes are appendicitis and ruptured duodenal ulcer; in women, gastric ulcer. Other causes are extension of hepatic abscess, empyema perforating the diaphragm, extension of kidney or pelvic abscess, and local tuberculous or (rarely) cancerous processes.

Diagnosis.—In a case of suspected abscess exploratory puncture may be performed. The needle should not penetrate beyond 3½ inches, so as to avoid puncturing the portal vein. In a right-sided *empyema* of the chest the upper border of the dulness, when continuous with that of the liver, is concave, being higher towards the spine. In *hepatic abscess* the liver is tender and enlarged below the costal margin, but it is often impossible to distinguish subphrenic from hepatic abscess. A variety containing air so greatly resembles pneumothorax that it is called *pyopneumothorax subphrenicus.* Exploratory needling, sometimes under chloroform, should be employed to complete the diagnosis. It is usually due to perforated gastric ulcer or abscess of the lung.

The *Prognosis* is fair if surgical treatment is carried out thoroughly and in time.

§ 271. **Actinomycosis of the Liver** is a condition which may be mistaken for abscess of the liver. It is due to the absorption of the ray fungus from the intestines, and starts as one or more foci in the liver substance, which slowly enlarge and may undergo suppuration, though the frequency of this latter is debated.

The *Symptoms* consist of vague uneasiness referable to the liver, with gradually increasing enlargement—at first uniform, later on unequal, the organ becoming prominent in one place. Exploration with trocar may yield no results ; but if the tumour is laid open, the characteristic greenish fluid with yellow specks is obtained in which the ray fungus is found, which clinches the diagnosis.

Distoma hepaticum rarely affects man. It causes painful enlargement of the liver, with pyrexia, jaundice, ascites and gastro-intestinal disturbance. The ova and flukes are found in the stools and vomited matter.

VI. *The illness has been ushered in by deep* JAUNDICE *and* PROFOUND CONSTITU-TIONAL SYMPTOMS ; *the liver dulness* DIMINISHES *rapidly.* The disorder is ACUTE YELLOW ATROPHY OF THE LIVER.

§ 272. **Acute Yellow Atrophy** (Malignant Jaundice, Icterus Gravis) is a rare disease characterised by intense jaundice and cerebral symptoms, extensive necrosis of the liver cells, with rapid diminution in volume of the organ, occurring especially in pregnant women, and usually ending fatally.

Symptoms.—(i.) The premonitory symptoms may be slight, resembling a catarrhal jaundice. There is increasing tenderness over the liver. (ii.) In a few days or weeks severe symptoms set in, with deepened jaundice, headache, and delirium, and the patient passes into the typhoid state. (iii.) Hæmorrhages occur from the stomach, bowel, and bladder, and there may be petechiæ under the skin. (iv.) Fever is usually absent during the course of the illness, but at the end it may be high. (v.) With the onset of the severe symptoms the liver dulness begins to rapidly diminish. The spleen is usually enlarged. (vi.) The urine is characteristically altered, containing bile and showing marked diminution in uric acid and urea, with increase of the ammonia coefficient and sometimes acetone. Leucin and tyrosin are sometimes found crystallising out on evaporating a few drops of urine on a slide (Fig. 70).

Diagnosis.—Acute Yellow Atrophy is not likely to be mistaken for any other liver disease after the acute symptoms set in. X-ray examination shows reduced size of

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[1] Mr. H. L. Barnard has described six clinical varieties, according to the position of the abscess (*Brit. Med. Journ.,* 1908, vol. i., pp. 205, 371, 429).

the liver. A similar fatal disease of the liver occurs in workers with trinitrotoluol. In phosphorus poisoning the liver is enlarged, and signs of irritant poisoning precede the onset of the jaundice.

Etiology.—Predisposing Causes.—(i.) Acute Yellow Atrophy is most common under middle age, though rare in children ; and (ii.) in women, especially during pregnancy. (iii.) Dissipation and excesses of any kind are said to predispose. *Exciting Causes.*— (1) It may be a complication of infective fevers, such as typhoid fever and influenza ; (2) it is found in delayed chloroform poisoning ; (3) it occurs in some cases of secondary syphilis. It is thought to be more frequent since the introduction of intensive treatment with arsenical preparations. In these cases the destruction of the liver cells may be due to the toxic effects of arsenic as well as those of the spirochæta pallida.

Prognosis.—The disease is very fatal. After the severe symptoms set in the patient usually dies in a comatose condition within a week. Pregnant women usually abort.

The *Treatment* is very unsatisfactory. During the preliminary stage the disease is treated as in catarrhal jaundice. Warm baths, diaphoretics, rest, milk food, large doses of bicarbonate of soda and glucose, and diuretics may be tried. In all cases of syphilis under treatment with arsenic preparations intravenously, the urine and if necessary the blood should be watched carefully for the presence of bile pigments, and the treatment intermitted if they are found.

CHRONIC DISEASES OF THE LIVER

§ 273. Routine Procedure.—It will be remembered (§ 262) in the physical examination of a patient suspected to be suffering from hepatic disease that the *first* and most important question to investigate is whether there is *any alteration in size*, especially enlargement *of the organ* (by palpation and percussion). (2) For reasons which will be apparent below, the question next in order of importance is whether there is any *pain or tenderness* in the organ. And then (3) is there any *jaundice?* (4) Is there any *ascites?* (5) In every case of suspected liver disease the spleen (§ 284), the stools, and the urine should be carefully examined.

The numerous *fallacies* in the alteration of the size of the liver dulness must be carefully studied (§ 260).

Classification.—By common consent chronic diseases of the liver are divided into those in which the AREA OF DULNESS IS NOT INCREASED, and those in which the AREA OF DULNESS IS INCREASED ; and these latter are grouped into painful and painless enlargements.

A. The organ is of **normal** or **diminished size** in—

B. The organ is **increased in size,**—

  a. Without pain or tenderness—

  b. With pain or tenderness—

A. In the first group, in which the liver is of **normal** or **diminished** size, there are only two disorders, I. FUNCTIONAL DERANGEMENTS; and II. ALCOHOLIC CIRRHOSIS; and these are two of the commonest hepatic disorders met with.

TABLE XIX. CHRONIC DISEASES OF THE LIVER.

	Size and Surface.	Pain.	Jaundice.	Ascites.
I ADVANCED CIRRHOSIS OF THE LIVER (*atrophic alcoholic cirrhosis*)	After enlargement it becomes DIMINISHED Surface irregular (hobnail)	None	Generally absent, never marked	A very prominent symptom
Ia HYPERTROPHIC CIRRHOSIS Of alcoholic or phthisic, or biliary origin	Enlargement may be very great Surface hard and may be nodular	Varies	Varies.	Usually absent
II CHRONIC CONGESTION .	Slight enlargement Surface smooth	Present, but slight	Slight	Usually some
III FATTY LIVER	Moderate enlargement Surface smooth	Absent	Absent	Absent
IV LARDACEOUS or Amyloid LIVER	Enlargement may be very great Surface smooth	Absent	Absent	Absent
V CANCER OF LIVER	Great enlargement Surface uneven	Severe	Usually present	Usually present
VI HYDATID LIVER (rare in this country)	Outline of dulness arched or distorted	Absent, unless near surface	Usually absent	Absent

1. *There is* **no alteration in the size of the liver,** *but the patient complains of* LETHARGY, *vague digestive disturbances, sleepiness after meals, furred indented tongue,* CONSTIPATION, *headaches, and there is a frequent deposit of* URATES IN THE URINE *on cooling.* There is probably FUNCTIONAL DERANGEMENT OF THE LIVER.

§ 274. **Functional Derangement of the Liver** [2] certainly constitutes one of the commonest of the minor ailments that affect a highly civilised community. Very careful percussion may perhaps detect slight enlargement, but generally, if there is any enlargement present, it indicates congestion, Active (§ 263), or Passive (§ 280).

Symptoms.—There are two manifestations of functional derangement which deserve special notice—constipation and lithuria. 1. The common complaint, " My liver is sluggish," is often equivalent to saying that the bowels do not act properly. Certainly, *constipation,* attended by pale-coloured fæces, due to a deficiency in the amount of contained bile, is a frequent accompaniment of disordered liver. The amount of bile in the stools is not, however, an absolute guide to the activity of the liver.

[1] The presence of jaundice and ascites depends on enlargement of the glands in the fissure, generally considerable in the later stages.

[2] The introductory remarks at the head of this chapter may well be perused in this connection.

Diarrhœa alternating with constipation, and flatus passed per rectum, may be present.

2. Excess of urates in the urine, which appear when the urine cools as a pink or orange deposit, is said to be evidence of defect in the liver function. The blood contains not necessarily uric acid, but partially elaborated substances belonging to the chemical series which connects purin bodies on the one hand and urea on the other. Purin bodies are found in certain foods (§ 464) and in the nuclei of cells. Even on a purin-free diet the urine contains about 0·2 gm. purin nitrogen. This is derived from the body tissues, especially the leucocytes and the muscles, and is increased when there is cellular activity and destruction after unaccustomed exercise, and when the liver action is defective. In febrile states the increased output of urates is probably due to increased leucocytic activity. When there is decrease in the quantity of the urine it contains uric acid and urates ; when there is more excretion the less highly organised purins are present—xanthin, hypoxanthin, also hippuric acid. Murchison long ago pointed out that excess of urates was met with in structural diseases of the liver, and in functional derangement. The so-called " lithæmia " is accompanied, according to the same authority, by a great variety of symptoms—depression of spirits, irritability, lethargy, a disinclination for work, aching pains in the limbs, vertigo, sleeplessness sometimes, undue drowsiness at others, dyspepsia, palpitation, irregularity of the pulse, and high blood-pressure, or sometimes enfeeblement of the circulation and general enfeeblement of the body.

3. " Functional derangement of the liver may exist for years without any other symptom than the frequent deposit of lithates, and occasionally lithic acid, in the urine. But if neglected it may ultimately be the means of developing *gout*, structural diseases of the liver and kidneys, or some other serious malady " (Murchison). 4. *Sugar in the Urine* —*i.e.*, temporary or permanent glycosuria (diabetes)—may possibly in some cases be regarded as a manifestation of functional derangement of the liver.

It may, however, be assumed that glycosuria might arise in one or more of three ways : (a) Imperfect glycogenesis in the liver, the sugar passing through the liver unchanged ; (b) increased conversion of glycogen into sugar, which results whenever the circulation through the liver is increased—*e.g.*, by vaso-motor paralysis of the hepatic artery ; and (c) diminished destruction of sugar in the blood or tissues.

Etiology.—Functional disorder of the liver and consequently the other symptoms above named may be *secondary* (a) to the continual over-functioning of the organ, (b) to diseases of the alimentary tract, (c) to diseases of the heart or lungs, and (d), as above mentioned, to pyrexia. When *primary*, its principal causes are—*Errors of diet*, especially rich, sweet, greasy foods, and alcoholic beverages, *i.e.*, indigestible and excessive food rather than food with purin bodies. Alcohol *combined with sugar* (*e.g.*, port and other fruity wines) is specially injurious ; or taken in the form of undiluted spirit, particularly on an empty stomach, is infinitely more harmful than dilute alcohol at meal-times. *Deficient supply of*

oxygen, such as deficient exercise, or confinement in ill-ventilated rooms. In spite of much recent research on purin bodies, but little can be added to the clinical observations noted by Murchison. The individual with the uric acid diathesis is one whose liver cannot deal with the purin bodies as can the normal liver.

Treatment.—(1) *Diet* is certainly the most important feature of the treatment. Avoid particularly alcohol, all highly-seasoned and rich foods, sweets, pastry and butter, because of their dyspeptic consequences. Murchison stated : " In most cases of lithæmia, a diet consisting chiefly of stale bread, plainly-cooked mutton, white fish, poultry, game, eggs, a moderate amount of vegetables, and weak tea, cocoa, or coffee answers best ; while in others the patient enjoys best health on a diet composed of milk, farinacea, vegetables, eggs, and occasionally fish." Haig's diet is still more vigorous,[1] and undoubtedly the quantity as well as the quality of the food must be regulated. There is no doubt that many sufferers take more food than can be dealt with by the liver. It is here that inherited peculiarities play such an important part, for what is too little food for one man may be too much for another. The mixture of much carbohydrate with proteid seems injurious to these individuals. Various dietaries are given in § 236. Whey is a diuretic and is apparently beneficial otherwise also. (2) Abundant exercise in the open air to supply the necessary oxygen is only second in importance to diet. (3) Hydragogue and cholagogue aperients—*e.g.*, the regular administration of salines (Hunyadi, Carlsbad, or Friedrichshall waters) every morning, and calomel once or twice a week (F. 46, 51, and 67). (4) Personally I have found bark or mineral acids (especially nitrohydrochloric) and bark, taken shortly before meals, very efficacious in some cases. (5) Among the other drugs chlorides, iodides, and bromides are recommended for the various conditions, as indicated by the symptoms. Opium is contra-indicated. If this treatment fails, turn to that of Acute Congestion (§ 263).

II. *The area of liver dulness is* **diminished,** *and if the surface can be felt it is* HARD AND UNEVEN (*hobnail*) ; ASCITES *is probably present, but no very distinct jaundice ; the spleen is enlarged, and the patient is subject to* HÆMORRHOIDS, *and* HÆMORRHAGES *from the stomach and bowel.* The disease is ATROPHIC ALCOHOLIC CIRRHOSIS.

§ 275. **Atrophic Cirrhosis of the Liver,** or, as it is sometimes called, Alcoholic Cirrhosis, Interstitial Fibrosis of the Liver, or Interstitial Hepatitis, consists of a progressive degeneration of the liver cells, with an increase of the interstitial fibrous tissue, leading to portal obstruction,

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[1] Dr. Alexander Haig's diet for uric-acidæmia consists of : Bread, 10 ounces ; oatmeal, 2 ounces ; milk, 2 pints ; cheese, 2 ounces, rice, 2 ounces, vegetables and fruit, 12 ounces. Vegetables, fruit and bread may exceed these quantities, but this observer maintained that the nearer a patient adheres to this dietary, the less likely is he to suffer from uric-acidæmia, gout, rheumatism, and allied diseases. These diseases he regarded as being largely dependent on faulty diet, and especially the consumption of animal food (proteids) in excess, and such as contain uric acid and its antecedents (*e.g.*, xanthin). The foods quite free from these substances are bread, macaroni, rice, and other cereals, potatoes, vegetables, nuts, and fruit.

and a shrinkage of the organ. Pathologists are now agreed that the interstitial fibrosis is secondary to the atrophic degeneration of the hepatic cells. Clinically there are two varieties of Alcoholic Cirrhosis—the *Atrophic* form, which is a very common condition; and the *Hypertrophic* form, which is relatively rare. The adjectives have reference *to the size of the organ*, for whereas the Atrophic form soon becomes diminished, the Hypertrophic form is enlarged throughout the disease. The hypertrophic form is further distinguished by a tendency to jaundice without ascites; and histologically the fibrosis has a uni-lobular distribution, instead of being multi-lobular as in atrophic cirrhosis.

Symptoms.—(1) In the early stage of the disease the organ may be enlarged, though rarely much so; but in the second and third stages the liver dulness is diminished. The liver is small and hard, and the surface is often nodulated, hence it is known as the " hobnail," or " gin-drinkers' " liver. There is a feeling of uneasiness and weight in the hepatic region. (2) The onset of the disease is very slow and insidious, extending sometimes over years. Gastric symptoms, such as *morning sickness*, and the other symptoms of alcoholic dyspepsia, are alone complained of for a considerable time. These are followed by symptoms of chronic gastritis, debility, and emaciation. The patient's aspect is very characteristic, with dilated *venous stigmata* in the cheeks. (3) Jaundice appears in the later stages of the malady in about one out of three cases. (4) Symptoms of portal obstruction occur (§ 261), and hæmatemesis is sometimes the first obvious symptom; the spleen becomes enlarged, and ascites (which is present in 80 per cent. of the cases) may be very considerable in amount. (5) In the concluding stages of this disease, when the secreting tissue of the liver is destroyed, the patient falls into a comatose state, with muttering delirium, which resembles uræmia and the typhoid state, except that there is pyrexia in the latter. This precise clinical resemblance is quite in keeping with the fact that the liver takes part in the elaboration of urea; so that when its cells are destroyed the blood becomes charged with a number of nitrogenous products, which cannot be eliminated.

Etiology.—(1) Cirrhosis of the liver is most common between thirty-five and sixty; it is rare under twenty-five. Men are much more frequently affected than women. (2) Alcohol is undoubtedly the most usual cause of atrophic cirrhosis, especially when taken in small quantities, frequently, or when taken *neat on an empty stomach*, the patient perhaps never becoming intoxicated. (3) Alcoholic excess is now known to be only one of the causes of cirrhosis of the liver. Syphilis is in some cases a predisposing factor, and so are many bacterial infections. (4) In poisoning by T.N.T. and tetrachlorethane the cirrhosis is subacute or even acute.

Diagnosis.—*Cancer* of the liver is only difficult to diagnose from cirrhosis in the early stages; but usually it runs a more rapid course, and is accompanied by more pain, and more intense jaundice. The spleen is not usually enlarged in cancer. In *passive congestion* of the liver with ascites there are evidences of a cause, such as heart or lung disease. In the absence

of ascites early cirrhosis may be mistaken for the other causes of liver enlargement. The enlargement of the spleen in atrophic cirrhosis may lead to the primary condition being overlooked. The liver is much reduced in size in starvation. *Chronic peritonitis* with effusion may not be recognised as such until the organs can be palpated after paracentesis.

Prognosis.—The disease has a slower and more insidious onset than hypertrophic cirrhosis (below), and is in most cases a more serious condition. If the patient is seen before signs of portal obstruction supervene much can be done, but if not until afterwards, the prognosis is grave. The outlook is more favourable in patients who are young (under thirty), and where the general health is good. *Untoward Symptoms.*—Although restoration to comparative health has occurred after the development of ascites, it remains true that, as a general rule, with the onset of rapid ascites the end is in view, the patient rarely living more than a few months. When there is rapid reaccumulation of fluid after paracentesis, and little benefit is derived from treatment, the course tends towards an early death. Pleurisy, renal disease, or peritonitis are occasional complications.

Treatment in the early stages is practically the same as that employed for chronic congestion of the liver, and chronic gastritis (§§ 280 and 233). The habits of the patient must be corrected, and the diet reduced to the simplest elements ; milk should be the staple diet in advanced cases. Alcohol must be completely cut off, and regular exercise taken. A course of salines should be ordered to be taken in the early morning, and rhubarb or mercurial pills at night. Ammonium chloride and iodide of potassium are valuable remedies in the stage of enlargement of the liver. If portal obstruction and ascites have set in, see § 209. The Mayo brothers attempt to influence the course of cirrhosis by splenectomy, which may cut off hypothetical toxins and certainly reduces the blood supply of the liver and so reduces congestion. Patients sometimes recover after repeated tappings, which gives time for the establishment of the collateral circulation. Surgical measures have been adopted for the artificial production of peritoneal adhesions for the establishment of the collateral circulation ; that usually adopted is " epiplopexy," or stitching the omentum to the anterior abdominal wall.

We now turn to those chronic liver diseases in which **the area of dulness is increased.** These may be divided into two groups—those WITHOUT PAIN AND TENDERNESS are described immediately below. If the enlargement is attended WITH PAIN AND TENDERNESS, turn to § 280.

There are four diseases with **enlargement** of the liver **without pain and tenderness** : I. HYPERTROPHIC CIRRHOSIS ; II. FATTY LIVER ; III. LARDACEOUS LIVER ; and IV. HYDATID and other rare diseases. In CATARRHAL JAUNDICE (§ 264), CHRONIC CHOLELITHIASIS, and some other disorders, the liver is somewhat enlarged, but this is not their main feature.

Other rare causes of PAINLESS ENLARGEMENT of the liver are chronic blood diseases, noticeably LEUKÆMIA and SPLENIC ANÆMIA, CHOLÆMIA

(§ 258), KALA-AZAR and MALARIA (p. 396). TUMOURS (§ 281) may not be accompanied by pain in the early stages.

I. *The liver is enlarged and* PAINLESS ; *its surface is hard,* JAUNDICE IS PRESENT, *but little or no ascites, and there is a long history of failing health.* The disease is probably HYPERTROPHIC CIRRHOSIS.

§ 276. **Hypertrophic Cirrhosis of the Liver** is a term employed in a generic or clinical sense to indicate a progressive enlargement of the liver due to an increase in the connective tissue of the organ with a tendency to jaundice. The condition may occur under at least five different aspects, due respectively to Alcoholism, Syphilis, Gall-stones, Chronic Heart disease, and Malaria. It may also be associated with Splenic Anæmia (then called Banti's disease). A rare variety of hypertrophic cirrhosis accompanied by pigmentation of the skin has been described under the name of hæmochromatosis. Glycosuria appears later ; hence the name " BRONZED DIABETES." The pigmentation differs from that of Addison's disease in that it avoids the oral mucous membrane and appears on parts exposed to light rather than to pressure and friction. The pigment contains iron (§ 451).

Ia HYPERTROPHIC BILIARY CIRRHOSIS (synonym : Hanot's disease).— In this form of cirrhosis the fibrous overgrowth occurs around single lobules, hence the name " uni-lobular cirrhosis." The disease is now classed as a distinct form of cirrhotic liver, in which the organ is enlarged *throughout the whole course* of the disease ; and there is a great tendency to jaundice, and but little ascites—features which contrast with those of the more common condition, Atrophic Alcoholic Cirrhosis, just described. *Symptoms.*—(1) The symptoms come on very insidiously, with a failure of the general health. The patient rarely applies for medical aid until (2) jaundice has set in, which may become very pronounced. The urine contains bile, but the stools retain their normal colour. (3) Fever occurs at intervals, and may be as high as 103° F. (4) In spite of the intense jaundice there are few or no signs of portal obstruction, and ascites is rarely, if ever, present. (5) The liver is uniformly, and may be considerably enlarged, hard, and sometimes rough. There is no tenderness and no actual pain (except during the feverish attacks), though a dull weight may be complained of in the hepatic region. The spleen is usually enlarged. (6) Hæmorrhages, purpura and telangiectases may occur. (7) A history of alcoholism is sometimes present, but in most cases the cause is obscure.

Diagnosis.—From *atrophic alcoholic cirrhosis* it is known by the absence of signs of portal obstruction, § 275. *Fatty* and *amyloid* livers are not accompanied by jaundice. *Cancer* has a more rapid and painful course. And see Table XIX, p. 390.

Prognosis.—Sometimes patients die within twelve months, with an acute onset of the typhoid state, but most live for a number of years, with signs of progressive emaciation. When occurring in children the general health may appear unaffected for long.

The *Treatment* of Congestion (§ 263) is applicable, according to the

predominating symptoms. Calomel, gr. $\frac{1}{10}$ to $\frac{1}{4}$ t.i.d. (0·006–0·016) for three days, with intervals of three days, continued for months has good results ; and drainage of the gall-bladder has cured some cases.

Ib. CARDIAC VALVULAR DISEASE results, as we have seen, in very considerable congestion of the liver. Long-continued passive engorgement of the liver gives rise to changes known as the " nutmeg liver," accompanied by more or less enlargement of the organ ; and this may be attended by a considerable degree of fibrosis. The diagnosis depends on the presence of cardiac valvular disease and other features (see Passive Congestion, § 280).

Ic. CHRONIC SYPHILITIC DISEASE of the liver generally takes the form of a diffuse hypertrophic fibrosis ; or it may be met with in the form of *gummata.* Undoubtedly, hepatic fibrosis may result from both hereditary and acquired [1] syphilis, though probably the gummatous form is commoner in the latter. In the inherited variety two forms of fibrosis occur. In one there is fine diffuse fibrosis between the individual cells (unicellular fibrosis) producing a uniformly smooth, firm liver ; in the other, coarse fibrosis with perihepatitis occurs, as in the acquired disease.

The *Symptoms* are variable. The liver is moderately enlarged ; there is not much tendency to jaundice and portal obstruction excepting in the final stages. There may be actual pain, especially when the capsule of the liver is involved ; but as a rule there are only indefinite sensations of illness, accompanied in the gummatous cases by a slight degree of intermittent pyrexia. In the gummatous form nodular projections may possibly be made out on the surface of the organ. The presence of such projections, accompanied by intermitting fever and a history of syphilis, in a young adult practically make the diagnosis certain. In the absence of a syphilitic history the occurrence of pain and local tenderness at intervals points to syphilitic rather than to alcoholic cirrhosis, because *perihepatitis and the involvement of the capsule* are prominent features of syphilitic cirrhosis. In the diagnosis from cancer we have mainly to rely on the efficacy of iodide, and the (usual) absence of jaundice and ascites in syphilitic disease. If ascites be present the Wasserman reaction is more marked with the ascitic fluid than with the blood.

The *Prognosis,* as a rule, is good, if the nature of the disease be discovered and it is treated by large enough doses of antisyphilitic remedies.

Id. CIRRHOSIS OF BILIARY OBSTRUCTION.—Hypertrophic cirrhosis has been produced experimentally in one half of the liver by ligature of one hepatic duct, and it is met with clinically in association with gall-stones, tumours or glands pressing on the bile-ducts. When acting as clinical clerk to the late Dr. Charles Murchison, I had the opportunity of observing a case of this kind occurring in a woman, aged forty-five, with a history of repeated attacks of biliary colic. There was great enlargement of the liver, with jaundice of three years' duration. The acholic stools aid the diagnosis of this form of hypertrophic cirrhosis.

Ie. MALARIAL CIRRHOSIS.—Many parasitic infections with chief incidence on the hæmopoietic system, *e.g.* malaria, kala-azar, histoplasmosis, harbour the causal parasite in the connective tissues of Glisson's capsule. The presence of the parasite gives rise to a fibroblastic reaction with enlargement of the liver. There thus arises a condition of hypertrophic cirrhosis, but it is seldom a prominent feature in any of these diseases, and it may be doubted if the newly-formed fibrous tissues ever retract enough to cause venous or biliary obstruction.

II.   *The enlargement of the liver is* PAINLESS *and uniform ; the surface*

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[1] I have met with several cases of marked diffuse fibrosis of the liver, due to acquired syphilis. Glisson's capsule was extremely thick, and large ramifying bands passed from it into the organ in all directions.

is smooth and soft ; there is NO JAUNDICE OR ASCITES, *and the* SPLEEN IS NOT ENLARGED ; *there is a history of alcoholism, or the patient is suffering from phthisis.* The disease is probably FATTY LIVER.

§ 277. **Fatty Liver** is a condition in which fat is deposited in the hepatic cells, commencing in the periphery of the lobules. It is nearly always associated with some other disease.

Symptoms.—(1) The liver is enlarged uniformly and is quite smooth. (2) Pain, jaundice, and portal obstruction are absent. (3) The accompanying symptoms are due to the cause of the fatty liver, and may consist, therefore, of debility, anæmia, etc. (4) The history of a *Cause* is important —viz., (i.) Chronic wasting disease, such as phthisis. (ii.) Fatty liver appears in association with fatty heart (*q.v.*) and general obesity. (iii.) It often occurs consequent on chronic alcoholism ; and a mixed degeneration of fat and fibrosis is not uncommon.

The *Diagnosis* from the painful enlargements of the liver is not difficult (see Table XIX). From *lardaceous* liver it is known by the absence of signs of lardaceous spleen or kidney, and by the absence of its cause.

The *Prognosis* and *Treatment* depend upon the primary disease —*i.e.*, the cause. It is hardly likely that the fat can be removed.

III. *The* **enlargement of the liver** *is* UNIFORM *and* PAINLESS ; *the surface is smooth and hard ; there is* NO JAUNDICE, NO ASCITES ; *the* SPLEEN IS ENLARGED ; *there is a history of prolonged purulent discharge, phthisis, or constitutional syphilis.* The disease is LARDACEOUS DEGENERATION.

§ 278. **Lardaceous (Amyloid or Waxy) Liver** is a condition in which the liver tissue is replaced by lardaceous material, which starts in the capillaries and smaller arteries of the organ, leading sometimes to an immense enlargement.

Symptoms.—(1) The liver is enlarged uniformly and smoothly, and feels firm and resisting ; (2) pain, jaundice, and portal obstruction are absent ; (3) the constitutional symptoms are due to the presence of the causal condition, and to the presence of amyloid disease of other organs.

Etiology.—(i.) Long suppuration and purulent discharge, as from necrosed bone ; (ii.) constitutional syphilis ; and (iii.) tubercular disease of the lungs or elsewhere. Amyloid liver has become much rarer since chronic suppurations have been obviated by improved surgical methods.

Diagnosis.—The presence or history of a cause renders the diagnosis of amyloid disease comparatively easy (see also Table XIX).

The *Prognosis* depends upon the amount of amyloid disease elsewhere. Diarrhœa, indicating amyloid changes in the intestines, abundant pale urine, with albuminuria, indicating amyloid disease of the kidneys, are untoward signs. If the cause is remediable, as by surgical treatment, the liver may decrease in size.

Treatment.—The indications are (i.) to remove the cause, and (ii.) to keep up the strength. The former is attained by administering potassium iodide in the case of syphilis, and by surgical treatment in the case of long-standing discharges. Tonics, such as iron and quinine with cod-liver oil, are useful.

IV. *The* **enlargement of the liver** *is* PAINLESS, *but* NOT UNIFORM, *and the upper margin of the liver dulness is perhaps* ARCHED ; *there is no jaundice or ascites and the spleen is not enlarged ; a thrill or vibration is felt on percussion.* The disease is HYDATID CYST.

§ 279. **Hydatid Tumour of the Liver** depends on the presence in the liver of a

parasite, rare in this country, though common in Australia, India, the Argentine, and Iceland, where dogs live in close association with man, and in Russia, where wolves are common.

Symptoms.—(i.) There is a slowly increasing enlargement of the liver, which is smooth, globular, and elastic, sometimes fluctuating. The right chest may be bulged outwards, with dulness in the axilla. When the fingers of the left hand are laid on the tumour and tapped with those of the right hand, the "hydatid fremitus," or "thrill," is felt in some cases. (ii.) Pain is absent unless the tumour is very near the surface, when great pain may be present, because *the capsule* is involved. (iii.) No constitutional symptoms appear unless the tumour presses upon the surrounding structures, or becomes inflamed and suppurates. (iv.) Any part of the body may be invaded, and the symptoms vary accordingly. The presence of multiple cysts in the abdomen gives rise to a very characteristic sensation on palpation ; it is compared to the sensation of palpating a bag of cricket balls. Jaundice may occasionally be caused by cysts lodging in the bile-ducts.

Etiology.—The parasite enters the alimentary canal of man by means of drinking water contaminated by fæces containing the ova of the tænia echinococcus, a tape-worm which may infect the dog. The embryo is carried to the liver, where it encysts and grows. The cyst so developed has a gelatinous wall, and contains a clear fluid. From the inner wall grow the tiny brood-capsules in which scolices or embryonic heads develop, each with a crown of characteristic hooklets.

Diagnosis.—*Abscess* of the liver produces pain and fever, and on aspiration yields grumous material like anchovy sauce. *Pleuritic effusion* on the right side, leading to dulness in the axilla, may resemble hydatid. In such cases a *bulging* outwards of the lower ribs over the liver points to
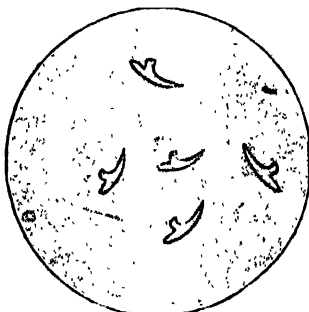
Fig. 72.—Hooklets from an HYDATID CYST in man ; the TÆNIA ECHINO-COCCUS.—Magnified about 150 times. These form the crown of hooklets around the anterior end of the scolex, and are absolutely distinctive of hydatid fluid. From a photomicrograph by Mr. Frederick Clark.

the presence of hydatid. A *renal cyst* has resonance in front, due to the colon. A history of residence in Australia, the Argentine, etc., should lead one to suspect hydatid in cases of slowly increasing enlargement of the liver, *with few other symptoms*. The symptoms of suppurating hydatid cyst of the liver are very like those of inflammation of the gall-bladder. Exploratory puncture is not justifiable, as it may set the scolices free, which subsequently form multiple cysts. X-rays are the most valuable method of diagnosis. The cyst fluid is pathognomonic. It is clear, opalescent, of low specific gravity, and contains a large excess of chlorides, no albumen (unless inflammation has taken place), and most characteristic of all—echinococcus hooklets (see Fig. 72). The blood shows eosinophilia, and the serum may give the complement fixation reaction with a suitable antigen.

Prognosis.—The patient may live for several years with no other symptoms than a slow increase in the size of the liver. The prognosis must be guarded even if the cyst, whose presence has been diagnosed, is safely removed ; for there may be other cysts present which will develop later. A cyst may remain quiescent for twelve years or more without dying or losing its potentiality for mischief. The cyst may suppurate, giving rise to the symptoms of liver abscess, or pyæmia may be set up. When a cyst leaks into the surrounding tissues, symptoms of acute poisoning occur—collapse, vomiting, and urticaria. Sometimes death occurs by the sudden rupture of the cyst into the pleura or peritoneum.

The *Treatment* is operative whenever possible. The cyst should be removed whole ; if this cannot be done, it should be incised and free drainage provided. This is

followed by the extrusion of the complete cyst wall in many cases. Every precaution must be taken against soiling the surrounding tissues; mopping out the cavity with formalin solution is recommended.

There are three diseases in which **enlargement of the liver** is attended with **pain and tenderness** : I. CHRONIC CONGESTION, II. CANCER OF THE LIVER, and III. ABSCESS OF THE LIVER. In CHRONIC CHOLELITHIASIS and several ACUTE DISORDERS the liver may be slightly enlarged and tender.

I. *The enlargement is moderate, smooth, and uniform,* PAINFUL, *and* TENDER ; *some jaundice and ascites may be present, the* SPLEEN IS ENLARGED, *and there are signs of congestion of the abdominal viscera.* The disease is probably CHRONIC CONGESTION OF THE LIVER.

§ 280. **Chronic Congestion** of the liver is a condition in which the enlargement is due to venous obstruction (passive congestion). Compare the opening remarks in § 263.

Symptoms.—(i.) The liver is tender, and a sensation of weight and fulness is complained of in the hepatic region. Expansile pulsation synchronous with the heart may be conveyed to the palpating hand in the early stages, but as the organ becomes firmer this is lost. (ii.) Signs of general venous obstruction appear. (iii.) Ascites develops, and the spleen is enlarged. The yellow discoloration of jaundice may arise. (iv.) Gastro-intestinal disturbances are common.

Etiology.—Passive congestion is the result of any backward pressure due to obstruction or failure of the circulation. In most cases this is caused by heart or lung disease, and especially mitral valvular disease. Any growth pressing on the inferior vena cava above the diaphragm has similar effects.

The *Diagnosis* is often aided by the recognition of the heart disease on which it depends. In some cases of *ascites* with anasarca of the legs, we may find both *hepatic enlargement* and *albuminuria*, and a difficulty may arise as to which was the primary cause of the condition—heart, liver, or renal disease. The difficulty is still further increased if extensive bronchitis prevents accurate auscultation of the heart. Now, in such cases, *the liver* may be excluded as the primary cause, if the dropsy in the legs clearly preceded the dropsy in the abdomen. The presence of hepatic enlargement is then a sign of great value as helping to exclude *renal* mischief, because enlargement of the liver is not a usual sequence of kidney disease, although it is a fairly constant result of *cardiac* valvular disease. In paroxysmal tachycardia the enlarged liver quickly decreases in size when the heart resumes its normal rate.

Prognosis.—The prognosis is altogether influenced by the cause of the congestion ; and the state of the heart is generally the measure upon which the patient's chance of a longer or shorter life depends. In mitral stenosis an enlarged liver with ascites is less grave than in mitral regurgitation, because it normally occurs at an earlier stage in stenosis. It is most serious in aortic disease, and especially regurgitation.

The *Treatment* is that of the cause, and our attention must be directed

to the heart and lungs.   Purgatives and light foods are necessary in order to relieve the strain on the portal system.   Leeches over the liver or bleeding may be indicated (see also Acute Congestion, § 263).

II. *The* **enlargement** *of the liver is* IRREGULAR ; *the* PAIN *and tenderness may be great ;* JAUNDICE *and* ASCITES *are present ; the spleen is not enlarged ; the patient is advanced in years, feeble and emaciated.*   The disease is CANCER OF THE LIVER.

§ 281. **Cancer** of the liver may be primary, but is usually secondary to disease elsewhere ; generally the stomach, rectum, or other part of the alimentary canal.

Symptoms.—(i.) Pain is an almost constant feature of cancer of the liver ; it is continuous, with exacerbations, and is independent, of food or position.   A certain amount of tenderness develops. (ii.) The enlargement of the liver is irregular, and nodules may be made out.   These are of a hard consistence, and increase rapidly.   There is also a less common diffuse form of cancer in which there are no nodules, and in which the liver is only slightly and uniformly enlarged.   In the nodular form the liver may be enormously enlarged. (iii.) Jaundice is usually present, *sooner or later,* and is intense and progressive.   An intense jaundice persisting over five to seven weeks in an old person should indeed always lead one to suspect cancer.   Ascites generally occurs either from involvement of the glands in the fissure, or the peritoneum.[1]   The spleen is not enlarged. (iv.) The general health of the patient is bad, and emaciation and cachexia may be present before any local signs are discovered.   Cancer may be present in another part of the body.   Fever occurs at intervals, especially in cases of primary cancer.

Causes.- Cancer occurs after middle life ; it is rare before thirty-five. It is liable to occur secondarily to cancer of the stomach or rectum.   When a patient has been the subject of gall-stones for a long period of time, cancer of the liver is apt to result in later life.

Diagnosis.—Jaundice is very rarely entirely absent in cases of cancer. This and the cachexia alone may justify a diagnosis of the condition. The diagnosis from *cirrhosis* may be difficult when nodular enlargement cannot be definitely made out, and when considerable ascites is present. In cirrhosis there is little or no pain and tenderness, the history of the illness is of longer duration, the spleen is enlarged, and the jaundice is not so intense.   The *inflammatory thickening* under the liver after a long history of gall-stones may resemble cancer, and can be distinguished only when time shows little or no increase in the enlargement.   *Syphilitic* liver has not so much pain and tenderness, is of slower growth, and rarely produces ascites.

Prognosis.—Cancer of the liver is usually fatal within six to twelve months, death taking place from exhaustion.   Untoward symptoms are

[1] Dr. Charles Murchison used to teach that *jaundice* with *ascites* in an *old person* usually indicated cancer.

rapid growth, ascites, or respiratory difficulties due to extension of the disease of the lungs.

Treatment can be palliative only. Morphia or opium is administered for the pain, and attention must be given to the relief of the symptoms of gastric distress, and to aid nutrition. With rest and care the patient may have periods during which the disease makes no progress, and which hold out to the patient false hopes of his ultimate recovery.

III. **Abscess of the Liver** also produces considerable hepatic enlargement, which is PAINFUL and TENDER. It has already been described among the Acute Diseases, § 244 ; but sometimes it runs a very chronic course.

§ 282. **Tumours of the Liver** other than CANCER (§ 281), HYDATID (§ 279), and GUMMA (§ 276, Ic.), are more rare. Their presence is manifested by *enlargement of the organ*, which may be regular or irregular, accompanied in some cases by constitutional symptoms. When, as in some cases of ACTINOMYCOSIS and DISTOMA HEPATICUM (§ 271), they assume an inflammatory form, pyrexia is present. SARCOMA OF THE LIVER is occasionally met with—*e.g.* Lympho-sarcoma—but it is most often secondary to deposits elsewhere, and the liver condition is only a subordinate part of the case. The patient may be younger than in the other form of malignant disease. Chondro-sarcoma, Melano-sarcoma, Tubercle, Angioma, Lymphadenoma, and Fibroma occur very rarely. Riedel's lobe is often mistaken for tumour (§ 212).

Floating Liver (*Dropping* or *Ptosis* of Liver, Hepatoptosis) is a somewhat rare condition due to laxity of the ligaments. It is apt to be mistaken for enlargement of the organ, and *vice versa.* The condition has been referred to under Abdominal Pain, because, if attended by symptoms, this is the principal one. There may also be vague neurasthenic symptoms.

THE SPLEEN

There is still some doubt as to the precise part which the spleen plays in the economy, and symptoms may be altogether wanting when it is diseased. Great diminution in size of the organ has been found *post-mortem* without any symptoms during life. When the spleen is removed surgically or rendered functionless by disease, its duties are assumed by the hæmolymph glands and the lymphatic glands. What the duties of the spleen may be are still matter for conjecture. The spleen does not appear to have an internal secretion as do the thyroid, suprarenal, and pituitary glands, but appears rather to suffer as the result of disease else-where. In embryonic life it is concerned with the formation of red and white blood corpuscles. In certain of the " blood diseases " in which it is enormously enlarged it resumes these functions. It is also largely con-cerned in the removal from the circulation of dead cells and of pigments, such as that of the parasites in malaria. It enlarges during digestion, and owns muscle fibres which give it the power of rhythmical contraction, the use of which is unknown, but in all probability the spleen is in some way necessary to the proper fulfilment of the digestive processes.

The spleen may be the seat of various congenital abnormalities. Of these the commonest is the presence of accessory spleens ; less common are multiple spleens and a multilobular organ.

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PART A. SYMPTOMATOLOGY

§ 283. In addition to the local pain and discomfort due to the enlarge-
ment of the organ, the symptoms which may arise include *extreme pallor*
of the skin, great *weakness*, and *alterations in the blood-cells*, chiefly leuco-
cytosis; but we are not sure that all of these are results of splenic disease.
Thus in "ague cake," for example, great enlargement takes place without
any symptom beyond the inconvenience due to the size of the organ. In
other instances a large spleen may, by simple pressure or by the formation
of adhesions, give rise to signs of disease in the neighbouring organs,
especially the stomach. Pain and local tenderness accompany acute
enlargements, and there may also be pyrexia and vomiting. The liver
and spleen are often enlarged together; one may preceded the other, or
both may be results of a common cause. The symptom which is found
to be most constantly associated with disease of the spleen is *anæmia*, the
various causes of which are discussed elsewhere (§ 431).

PART B. PHYSICAL EXAMINATION

§ 284. The only physical sign which can be relied upon as diagnostic
of splenic disease is enlargement of the organ, and this is most readily
made out by **Palpation.** When the spleen is enlarged, the anterior edge
of the organ, being free, makes its way downwards and forwards towards
the umbilicus. The *notch* in the anterior border is so characteristic that
it forms a strong point in diagnosis of any splenic tumour. METHOD.—
Stand on the right side of the patient, who should be lying on his back.
Pass the left hand across the abdomen, and lay it posteriorly over the
eleventh rib on the left side, and place the right hand flat upon the
anterior surface of the abdomen, with the tips of the fingers just below
the eleventh rib. By gently dipping them down into the abdomen, and
tilting the organ upwards with the left hand during inspiration, the splenic
notch may be felt if the organ is enlarged. It is more readily palpated
when the patient draws a deep breath. Normally, the spleen cannot be
detected by palpation, and even slight enlargements may not always be
appreciable. An enlarged spleen always has a space between its posterior
edge and the erector spinæ behind, into which the fingers can be dipped—
at any rate, in spare subjects. *Fallacies.*—Without being enlarged, the
spleen is readily palpable when it is displaced downwards, or is "floating."
It is sometimes displaced downwards in cases of deformed chest (*e.g.*,
rickets), large pleuritic effusions, and emphysema.

§ 285. The **Percussion** of the spleen is attended with some difficulty.
The organ is situated in the left hypochondrium, between the upper border
of the ninth rib and the lower border of the eleventh; and roughly between
the mid-axillary and scapular lines (Fig. 36, § 75). It extends obliquely
forwards and downwards nearly to the costal margin. It lies wholly
beneath the ribs, and the upper third is overlapped by the lung. Per-
cussion does not afford a very accurate means of investigation, but it is

well to remember that *a straight line drawn from the centre of the left axilla, obliquely downwards and forwards to the umbilicus, should be resonant in its entire length* (Gairdner's line). The spleen normally lies altogether behind this line, but if it be enlarged this line is impinged upon by dulness at the junction of its middle and lower thirds.

The SURFACE LANDMARK of the spleen may be said to form an oval, lying *obliquely* between the post-axillary and mid-axillary lines, and having, for purposes of description, four borders. The procedure for percussing out the *anterior* and *lower* borders differs from that used to elicit the *upper* and *posterior*, because the latter recede from the surface, the lung intervening. It is best to percuss at the end of an expiration, because the spleen is then less covered by lung. As mentioned above, palpation is preferable, but to define the *anterior* and *lower* limits by percussion the patient should lie on his back. (1) *Anterior border*—percuss *lightly* along the tenth rib, starting at its anterior end, and the note will be found to become dull about the *mid-axillary* line. (2) For the *lower border* percuss, also lightly, along the posterior-axillary line from below upwards, and the lower border should be reached about the lower edge of the eleventh rib. (3) To define the *upper* and *posterior* limits is very much more difficult, and very often—in fat subjects for instance—it is impossible. Fortunately, it is not of so much importance to map out the posterior border. The patient must either sit up or lie in a semi-prone position, resting on his right scapula. If he turns completely on to his right side, the spleen may fall away from the left side. His left hand should be placed on his head. *Upper border*—percuss with a *heavy* stroke just behind the post-axillary line, starting from the angle of the scapula and working vertically downwards. After repeating this several times, it will be noticed that the pulmonary resonance is impaired at the upper border of the ninth rib. (4) *Posterior border*—similarly with heavy percussion, by starting over the neck of the tenth rib and continuing along that rib anteriorly, you may elicit a change of note just in front of the scapular line.

Fallacies.—The dulness of *splenic enlargement* may be simulated by pleuritic effusion or consolidation of the left lung. The area of splenic dulness may be *diminished* by emphysema of the lungs, or distension of the stomach or the colon by gas. The splenic dulness may be altogether absent when there is a wandering spleen, or congenital absence of the organ.

§ 286. SPLENIC ENLARGEMENTS have three chief characteristics: (1) The splenic *notch* is felt on its anterior border; (2) the mass moves with respiration if not bound down by adhesions; (3) it is dull to percussion because the resonant colon does not lie in front of splenic tumours, as it does in front of renal tumours, Gairdner's line of percussion resonance (*vide supra*) being thus impinged upon. (4) When an area of dulness is due to splenic enlargement, its outline *resembles in shape* that of the normal spleen. (5) It is distinguished from neoplasms of the peritoneum, stomach, intestines, etc., by its smooth and firm surface. Irregular enlargements of the spleen are rare, and can only be diagnosed after careful examination has excluded disease of other viscera.

Splenic enlargements or tumours may have to be diagnosed from the following conditions: (1) *Renal tumours*, and especially movable kidney, in which there is resonant intestine in front of the tumour, and absence of resonance in the flank; (2) *enlargement of the left lobe of the liver*, in which the dulness is continuous with that of the right lobe, whereas splenic dulness rarely reaches to the middle line; (2) *cancer of the cardiac end of the stomach*, in which the dulness is less absolute, and there is "coffee-

ground " vomiting, etc., and the splenic notch is absent; (4) _ovarian tumour_, which (i.) will have grown from below upwards, (ii.) the hand cannot be pushed between the tumour and the pelvic brim as it can in the case of a splenic tumour, and (iii.) can be felt on vaginal examination; (5) _accumulation of fæces_, in which (i.) the tumour has an irregular outline, (ii.) doughy consistence, and (iii.) a course of purgatives and enemata will remove it; (6) _post-peritoneal tumour_, in which (i.) there is no notch, and (ii.) no resonance it; (7) _abdominal aneurysm_, when of sufficient size to be mistaken for the spleen, is attended by pain in the back, and evident expansile pulsation; (8) _deep-seated abscess in the abdominal parietes_ is tender, has a vague irregular outline, and is situated more superficially than a splenic tumour. In (9) _cancer of the splenic flexure_ of the colon the mass varies from day to day and there will be intestinal symptoms; (10) _pancreatic_ and _suprarenal_ tumours and _perinephric abscess_ may give rise to difficulty. (11) Rare causes of error are localised tubercular masses and the thickened colon of bilharziasis.

PART C. DISEASES OF THE SPLEEN

§ 287. The diseases of the spleen are all—if we except the relatively rare cases of wandering spleen and atrophy—comprised under the causes of **enlargement of the organ**, and its **diagnosis** therefore becomes a matter of considerable importance. Enlargement is detected by palpation aided by percussion as above mentioned. The mechanical effects of pressure, when the spleen is very much enlarged, are mainly dyspnœa and gastro-intestinal disturbance. These may be aggravated by attacks of peri-splenitis, with acute pain locally, vomiting, pyrexia, and sometimes diarrhœa. Œdema of the base of the left lung is not uncommon.

The **Causes of Enlargement of the Spleen** are most readily differentiated according as they depend upon or are associated with the following:

I. Acute infections.
II. Chronic infections.
III. Portal obstruction or congestion.
IV. Blood diseases.

V. Parasitic and tropical diseases.
VI. Infancy and childhood.
VII. Irregularity of the surface of the spleen.

Method of Procedure.—As pointed out in Part A., it is rarely that advice is sought for symptoms directly pointing to a splenic origin. Frequently the spleen is found to be enlarged when the patient is being examined for disease elsewhere. It should be remembered that in some obscure maladies the detection of an enlarged spleen may be an important clue to the diagnosis.

Inquiry should be made as to the HISTORY. Thus residence abroad suggests malaria; prolonged suppuration, lardaceous disease; fever and rigors, the presence of some pyæmic cause.

The AGE of the patient is important (see VI. below); in childhood certain conditions are common which are rare in adults.

The TEMPERATURE aids the diagnosis of certain infections.

EXAMINATION OF OTHER ORGANS may render the diagnosis easy. The condition of the LIVER is of especial significance in several diseases. Thus a large liver, jaundice,

and a normal spleen point to gall-stones or cancer, but if the spleen as well as the liver is large, these symptoms suggest cirrhosis or other obstruction. A very enlarged spleen with but slightly enlarged liver suggests some of the "blood diseases" which can be accurately differentiated only by an EXAMINATION OF THE BLOOD.

I. **Acute Infections.**—Almost all acute infections are apt to be accompanied by slight enlargement of the spleen, and as far as the acute specific fevers are concerned this is usually of little clinical significance. The enlargement is specially found with typhoid and typhus fever, and pneumonia. Sometimes, and particularly in TYPHOID fever, a splenic abscess may complicate the original condition. In such a case local symptoms of tenderness and pain will draw attention to the spleen. Again, these symptoms may arise in the course of some slight systemic infection, and be due to suppuration supervening in the area affected by an EMBOLISM or in some pre-existing cyst or tumour. Embolism due to cardiac disease causes (i.) acute sudden pain, and (ii.) local tenderness due to perisplenitis. Embolism due to pyæmia is usually known by the presence of the causal condition. In such diseases as leukæmia, in which the massive enlargement of the spleen is a prominent feature, the organ is liable to attacks of ACUTE CAPSULITIS, which may give rise to difficulty in diagnosis unless the possibility of their presence is borne in mind. A friction rub, due to localised peritonitis, may be audible during the acute attacks.

The diagnosis of the cause may be very difficult, and the most accurate balancing of probabilities may fail to reveal the truth. Expectant treatment is then to be adopted, and consists of hot applications to the spleen, rest in bed, and attention to the bowels. If the attack does not subside and the local signs become worse, the advisability of surgical interference must be considered. Fortunately this is rarely needed, and the attacks tend to resolve in a few days, leaving adhesions which may lead to trouble later (§ 283).

II. **Chronic Infections.**—(1) MALIGNANT or ULCERATIVE ENDOCARDITIS (§ 46) may give rise to embolism, which causes acute symptoms, or to a more chronic enlargement not wholly due to congestion, and difficult of explanation. The symptoms in the latter case may be exactly similar to those of splenic anæmia (§ 439), and may, moreover, occur when there is not the least suspicion of cardiac trouble. The importance of this lies in the fact that it is possible to remove the spleen with advantage to the patient in splenic anæmia, but the operation would usually be inadvisable in endocarditis. ABSCESS of the spleen may also occur in the course of this disease.

(2) SYPHILIS may cause a uniform enlargement of the spleen in the early stages of the toxæmia. Later, both spleen and liver may become enlarged, and the diagnosis may be very difficult. Ascites may supervene, and anæmia. If at the same time a degree of pyrexia be present for which no cause can be found, syphilis is probably in operation.

(3) TUBERCULOSIS may occur as miliary tubercle, as an abscess, as a capsulitis, or even as multiple tuberculomata. In no case is it likely to

be diagnosed apart from the existence of tuberculosis elsewhere. It is rarely primary in the spleen, and is then an exception to the rule; if diagnosed it might be operated upon. In some cases of splenic tuberculosis there is a marked polycythæmia instead of the anæmia which usually accompanies tuberculosis.

(4) LARDACEOUS (Amyloid) disease of the spleen is becoming rarer every year. It is known by: (i.) There is usually a history of syphilis, phthisis, or of chronic purulent discharge; (ii.) the liver shows signs of lardaceous disease, and diarrhœa may be present, due to involvement of the intestines; (iii.) the spleen may be very large, much larger than is commonly the case in acute or chronic infections.

(5) In the absence of fuller knowledge, HANOT'S DISEASE (BILIARY CIRRHOSIS) may come under this heading. The spleen may be enlarged before the liver in some cases. The diagnostic signs are considered in § 276.

(6) CHRONIC SEPTIC SPLENOMEGALY resembles Splenic Anæmia except in that it may present a leucocytosis. It occurs in many cases of sepsis, e.g., of the teeth or bowel. It is especially common in tropical climates after dysentery or other intestinal disorders. The prognosis is good if the causal sepsis can be eradicated. It does not as a rule lead to hæmatemesis. It has been called "Egyptian Splenomegaly," and has often been described in connection with malignant endocarditis. The importance of the condition resides in the fact that it is liable to be confused with Splenic Anæmia and a bad prognosis given in consequence.

III. **Portal Obstruction or Congestion.**—Any cause of portal obstruction, of whatever degree, will naturally lead to congestion in the whole of the splanchnic area, and in this the spleen will share. Thus the spleen is enlarged in (1) CARDIAC and CHRONIC LUNG DISEASE, with backward pressure in the venous system. The obstruction may be more absolute, as in (2) THROMBOSIS of the INFERIOR VENA CAVA. In this case the enlargement of the spleen may reach a greater degree than in congestive conditions of the liver, and where the thrombosis affects only the splenic vein the hypertrophy may be extreme, and the symptoms conform to those of splenic anæmia (of which, according to some authorities, it is the chief cause) (§ 439). (3) CIRRHOSIS of the LIVER (§ 275) is associated with splenic hypertrophy. (4) In SYPHILITIC fibrosis, however, the liver and spleen are usually simultaneously affected. (5) One cause of splenic congestion and hypertrophy must be mentioned, although of great rarity —viz., TORSION of the splenic pedicle. This is only likely to occur when the spleen is displaced by its increased weight (in splenomegaly), or when it is possessed of an unusually long pedicle (as in splenoptosis and wandering spleen). It is unlikely to be diagnosed except by operation.

§ 288. IV. "**Blood Diseases**," or diseases of myeloid and lymphatic tissue. This heading includes all those commonly known as "blood diseases." They merit individual remark, but for full descriptions the

reader is referred to other paragraphs. In almost all of these the acute attacks of capsulitis above mentioned are apt to occur.

(1) PERNICIOUS ANÆMIA is very often and (2) CHLOROSIS not infrequently associated with slight enlargement of the spleen. This never reaches a large size, and the fact of its doing so would be a sign that the diagnosis requires revision.

(3) In SPLENO-MEDULLARY LEUKÆMIA (§ 437) the spleen is characteristically enormous, but it is to be remembered that in LYMPHATIC LEUKÆMIA and in (4) CHLOROMA it may be just as large, even reaching 2 or 3 inches to the right of the middle line. In the latter diseases some degree of enlargement is almost invariable, and in the acute cases is always present. These diseases are diagnosed largely by the blood examination.

(5) HODGKIN'S DISEASE (§ 438) is known by : (i.) One or more groups of enlarged lymphatic glands are present ; (ii.) the splenic enlargement is slight.   •

(6) SPLENIC ANÆMIA (§ 439) could hardly be diagnosed without the enlargement of the spleen, which usually reaches very considerable proportions. As will be gathered from the remarks made above, this disease is no doubt destined to be subdivided into several groups when further knowledge is available. In the tropics it may be simulated by kala-azar and other diseases. There is a form of splenic anæmia which is found particularly in infants, and which has a tendency to occur in twins. In this the prognosis is better than in the adult form, and there are blood changes which serve to diffcrentiate it.

(7) ACHOLURIC JAUNDICE or cholæmia is associated with great enlargement of the spleen in the majority of cases. It is known by : (i.) it is often a disease of family incidence, (ii.) the presence of jaundice, and (iii.) the characteristic blood changes (§ 258).

(8) ERYTHRÆMIA is diagnosed by (i.) polycythæmia, which may reach a very high degree, and (ii.) the cyanosis, weakness, and parœsthesias to which it gives rise (§ 29).

V. Tropical Diseases.—The two most often met with are MALARIA and KALA-AZAR. In acute malaria the enlargement is not very great, but after many attacks it may be enormous without giving rise to much inconvenience. A history of attacks of ague occurring in a person who has been abroad leads one to suspect the cause of the splenic enlargement ; but the diagnosis is made only by finding the parasite in the blood. In kala-azar the spleen usually reaches a large size, and is rendered the more prominent by the emaciation of the subject. The diagnosis rests on the discovery of the parasite in the blood (rarely) or in the material obtained by liver or spleen puncture.

Rarer parasitic causes are HISTOPLASMOSIS, TOXOPLASMOSIS, and KREMPF'S SPLENOMEGALY (due to hæmogregarinæ hominis).

VI. In Infancy and Childhood RICKETS (§ 478) is one of the most frequent causes of slight enlargement of the spleen which may depend on intercurrent catarrhs of mucous membranes. In children the spleen

enlarges much more readily than in adults, and for reasons which would not be held adequate were an older person concerned. Congenital SYPHILIS and TUBERCLE are also more likely to be present in children, and are recognised by signs of the disease elsewhere; in syphilis the liver also is enlarged. There is a special form of splenomegaly associated with ANÆMIA in infants (§ 444), which has been mentioned above, and there is also a special form of KALA-AZAR in infants. In cyanosis from CONGENITAL HEART DISEASE there is sometimes marked enlargement of the spleen. Congenital ERYTHRÆMIA is also described.

VII. **Irregularity** of the surface of the enlarged spleen. This group includes quite a different class of disease to those above mentioned. The most important cause of enlargement is sarcoma, for there is some hope of cure if the spleen be removed early enough. It is rare, and usually occurs in children or young adults. It can only be diagnosed by exclusion. Other new growths are even more rare, and include lymph-angioma, fibroma, pulsating angioma (which may give rise to suspicions of aneurysm), secondary cancer, and cysts such as dermoids.

HYDATID cyst in the spleen may be diagnosed by (i.) the presence of marked eosinophilia in a person who (ii.) has resided in an affected country, (iii.) the serum reaction, and (iv.) sometimes by the presence of cysts elsewhere; (v.) the cyst may present the characteristic thrill on palpation.

LYMPHADENOMA may give rise to irregular enlargement, and certain congenital malformations are irregular.

The *Treatment* and *Prognosis* of splenic enlargement depend, for the most part, on the primary condition. The treatment of lardaceous disease and of hydatid is given under Hepatic Disorders (§§ 278, 279). The treatment of " Ague Cake " consists of (i.) removal to a non-malarious district, and the administration of quinine and tonics, with free saline purgation; (ii.) unguentum hydrargyri iodidi dilutum, rubbed over the splenic area, is a remedy which may be of value. (iii.) Violent movement must be forbidden, as the spleen may rupture. In chronic syphilitic splenomegaly splen-ectomy has been of use.

§ **289. Wandering Spleen** (Floating, Dropped, or Dislocated Spleen, Splenoptosis) may be readily mistaken for enlargement of that organ when met with in the lesser degrees of displacement. But when the dislocation is, as generally happens, consider-able, it is more often taken for a floating kidney. However, the presence of the notch, the fact that it can be made to recede upwards and that it comes down in front of the colon, aid in the diagnosis. The condition is mostly met with in multipara with pendulous abdomens. It may be accompanied by nervous symptoms, though these are less constant than in dislocation of some of the other viscera. If troublesome, the condition may be relieved by removal of the organ, an operation which has been performed several times with good results.

Atrophy of the Spleen is, as a rule, unattended by symptoms. It is, as Bristowe said, a condition not infrequently met with. It may be congenital, but its com-monest causes are: I. CIRRHOSIS of the spleen, due to an increase in the interstitial tissue, the result, as in cirrhosis of the liver, of alcohol; and II. CONTRACTION OF THE FIBROUS CAPSULE, usually of syphilitic origin. The syphilitic deposits in the capsule of the spleen sometimes take on a cartilaginous change, and form plates of cartilage. I have come upon them several times in the dead-house, but they had been unattended by symptoms during life. I have also met with some five cases of marked atrophic condition of the spleen, without symptoms during life, death having occurred from independent causes.

§ **290.** The following are the indications for SPLENECTOMY: (i.) Rupture of the spleen, torsion or similar acute emergency. These constitute an absolute indication

in all cases in which the patient is likely to survive the operation. (ii.) Tumours or abscess of the spleen which cannot be dealt with by less radical measures. (iii.) Persistent attacks of peri-splenitis or other disabilities directly dependent on the size and weight of the organ. These are a sufficient indication in nearly all cases but do not allow of operation in leukæmia or erythræmia. (iv.) Cases in which splenectomy has been shown to exercise distinct benefit on the general disease present : e.g., early cases of splenic anæmia, chronic infective splenomegaly and acholuric jaundice. Cases of pernicious anæmia are not certainly benefited except for a short time, nor does Hodgkin's disease fall within this group. (v.) Cases in which the circulation through the liver is embarrassed as in cirrhosis, and in which diminution of the volume of blood passing through the portal vein may be of advantage. Banti's syndrome falls in this group, as well as a proportion of anomalous cases described as Hanot's cirrhosis.

Of the blood diseases only two—splenic Anæmia or Chronic Infective Splenomegaly, and Acholuric Jaundice—demand treatment by splenectomy. Even so, splenectomy is to be undertaken only when it is evident that the disease is impairing, or likely to impair the general health and working ability of the patient. Then the operation should be done early, and particularly if there has been even a single attack of peri-splenitis. Peri-splenitis causes adhesions and one attack will certainly be followed by others. What is, in skilled hands, a fairly simple and safe operation will then become extremely difficult and dangerous and the patient should not be allowed to drift on in ignorance of this fact. In no case should a patient be submitted to operation without adequate preparation, by transfusion if necessary. Pneumonia at the left base is especially to be guarded against after operation—by deep breathing, by avoiding constriction of the chest with bandages, etc.

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CHAPTER XIII

THE URINE

THE intricate subject of renal diseases is rendered more comprehensible by a brief consideration of their history. In 1812 Wells [1] found that albumen in the urine was associated with certain forms of dropsy. It was not, however, until 1836 that Bright [2] went a step further and discovered that dropsy and albuminuria, when associated together (in the absence of heart disease), were indicative of disease of the kidneys. The term " Bright's Disease " became synonymous with inflammatory, non-suppurative disease of the kidneys. We now know that many disorders of the kidney present dropsy and albuminuria at some stage of their course. Thus, for example, these symptoms may be caused by the toxins of diphtheria, scarlet fever, measles, pregnancy and certain poisons, and possibly by the organisms of pneumonia, coli, typhoid and other diseases, entering the kidney substance by the blood or ascending by the urinary passage. With amyloid disease and the degenerative conditions due to age, alcohol, lead, etc., there is often associated a certain degree of inflammation of the kidney substance. The term Bright's disease has been loosely used in connection with several forms of nephritis. In acute nephritis the inflammatory condition attacks chiefly the glomerulo-tubular epithelium ; in chronic nephritis the disease may affect the tubules or the interstitial tissue of the organ. One form of chronic glomerulo-tubular nephritis (chronic interstitial nephritis) is very often associated with a widespread cardio-vascular change in which high blood-pressure and its general symptoms are more pronounced than the urinary symptoms. By degrees the term Chronic Bright's Disease has come to be applied to cases in which the chief and perhaps the only symptoms are cardio-vascular. Even although it was shown that high blood-pressure and cardio-vascular changes may occur without renal disease,[3] the term Chronic Bright's

[1] Wells, Transactions of the Society for the Improvement of Medical and Surgical Knowledge, iii., 194, London, 1812.
[2] Bright, Guy's Hosp. Rep., I, London, 1836.
[3] The author's investigations into the large series of renal cases on the one hand and cardio-vascular cases on the other which presented themselves at the Paddington Infirmary revealed this (Trans. Med. Soc., London, 1897, 1898 ; the Lancet, 1897, vol. i., pp. 882 and 1235 ; Brit. Med. Journ., January 23, 1897 ; and Trans. Path. Soc., London, 1904).

Disease is still sometimes loosely and incorrectly applied to high arterial pressure and its associated symptoms.

Renal diseases are for the most part chronic, and often obscure ; but, with a knowledge of these historical data, their study would be comparatively simple were it not for another confusion which has arisen owing to the numerous terms applied by pathologists to diseases which differ anatomically, though not always clinically. In what follows we shall be concerned only with the clinical aspect of renal disease.

It is not always possible in practice to separate kidney diseases proper from disorders of other parts of the urinary tract, because changes in the urine are common to them all. It will be necessary, therefore, to refer to disorders of the bladder, prostate, and urethra for diagnostic purposes, though their treatment comes mainly within the province of the surgeon.

PART A. SYMPTOMATOLOGY

§ 291. The chief function of the kidneys is the elimination of nitrogenous waste. When this is interfered with by structural or functional disease, a toxic condition results, which is known as uræmia. Other functions are the removal of acid products, the maintenance of the blood sugar level and of the best salt concentration in the tissues and fluids.

As a consequence of the deep-seated position of these organs, the local symptoms referable to the kidney are, except in cases of Tumour or displacement, of subordinate importance. The most constant and CARDINAL SYMPTOM of kidney disorders is some **Alteration in the Urine**, which, as an indication of renal disease, corresponds to the physical signs in other organs, and is dealt with in PART B. of this chapter. The cardinal symptoms next in order of importance are **Pallor of the Surface** and **Dropsy**. General Symptoms, due to toxæmia resulting from the retention of nitrogenous wasting, also accompany these diseases.

Pallor of the Surface and Malaise are very constant features of all organic kidney diseases. To the experienced eye the pallor differs from that of anæmia in a manner somewhat difficult to describe. The skin has a " waxy " hue, a simile which is still further exemplified when dropsy is present. It affects the whole body, but is always most evident in the face. In chronic interstitial nephritis the pallor has a greyish hue. The diagnosis from other causes of pallor will be found in Chapter XVI, § 431.

§ 292. **Renal Dropsy** is of *general* distribution, in which respect it differs from cardiac dropsy, which starts in the *legs* or most dependent parts, and from hepatic dropsy, which starts in the *abdomen*. It is, however, most evident in the loose cellular tissue—*e.g.*, around the eyelids, where it is most marked on first rising in the morning. Towards evening the ankles become œdematous, or, as the patient may express it, a " ridge is present around the top of the boot." In severe cases (*e.g.*, in acute

nephritis) the eyes may be almost closed by the swollen lids, and at the same time there may be signs of dropsy in the serous cavities—the pleura, peritoneum, and pericardium. Œdema of the solid organs also occurs in . severe cases, and death may be produced by pulmonary œdema. Œdema glottidis is another serious though less frequent complication.

Dropsy is by no means an equally constant feature in all diseases of the kidney. In *acute and chronic parenchymatous nephritis* (*i.e.*, disease in which the renal epithelium is primarily affected) dropsy is almost invariably present. But in *chronic interstitial nephritis* and *lardaceous kidney* it is comparatively rare ; in the former it may occur late in the course of the disease, when it is generally due either to cardiac failure or to secondary inflammation of the renal epithelium. In uncomplicated *pyelitis* and *neoplasms* dropsy is not present.

§ 293. A large number of **General Symptoms** occur consequent on the retention of the nitrogenous waste products : Cardio-vascular changes, hæmorrhages, breathlessness, affections of the nervous system, ocular changes, gastro-intestinal disorders, and secondary inflammations.

CARDIO-VASCULAR CHANGES frequently accompany renal disease. In acute and chronic renal disease there is usually high blood-pressure, and often dilatation of the heart. The accentuated second aortic sound which accompanies this high pressure is a useful indication in some cases for bleeding, or other measures for the reduction of pressure. In chronic interstitial nephritis the high pressure is apt to be followed by a thickening of the arteries due to hypertrophy of the muscular coat.[1] The left ventricle becomes hypertrophied, and, in the later stages, signs of cardiac dilatation and failure may ensue.

HÆMORRHAGES sometimes occur in chronic renal disease, a consequence of the high pressure, combined in most cases with a diseased state of the blood-vessels. Epistaxis, for instance, may be the first symptom which leads to the discovery of chronic renal disease. Bleeding from the stomach or intestines, and purpura, sometimes occur. *Cerebral hæmorrhage is the most frequent cause of death.*

BREATHLESSNESS, apart from that due to chronic pulmonary œdema, is a common accompaniment of renal disease. Renal disease is the most frequent cause of acute pulmonary œdema (§ 97). A paroxysmal dyspnœa, coming on during the night in a person of advanced life, should lead us to suspect the existence of chronic renal disease, even although the patient may continue his occupation. *Cheyne-Stokes respiration* may develop towards the end, with or without other symptoms of uræmia.

NERVOUS SYMPTOMS are not infrequent, apart from the cerebral hæmorrhage just referred to. Thus, *headache* is a symptom which accompanies all renal diseases, particularly those forms which terminate in uræmia. Experience among the aged shows that chronic interstitial nephritis is one of the most frequent causes of headache in advanced life. The patient may continue his work, and present no other symptom, but an examination of the urine may reveal the existence of chronic renal disease. *Vertigo, tinnitus*, and various *neuralgias* may also be complained of. *Insomnia* in the aged is another common symptom of chronic renal disease. The patient complains that he readily drops off to sleep, but as readily awakes, and that he may do so a

[1] See also Arterial Hypermyotrophy, § 79. With certain methods of preservation and hardening, or with insufficient staining, the middle coat presents precisely the appearance of fibrous tissue ; but well-stained logwood preparations always reveal the rod-shaped nuclei, and acid orcein will always reveal the elastic tissue boundaries of the tunica media.

dozen times every night. As the uræmic condition increases, however, *drowsiness* supervenes, which may pass into *coma*, without or without muttering *delirium*. Sometimes *convulsions* occur before death.

OCULAR CHANGES frequently accompany renal disease attended by albuminuria ; and so characteristic are the changes that albuminuria may be diagnosed by their presence. Typical alterations occur in the fundi, with resulting loss of visual acuity —œdema and swelling of the retina, papillitis, flame-shaped hæmorrhages, and white spots of fatty degeneration. Changes in the arteries may also be seen.

GASTRO-INTESTINAL symptoms attend some renal diseases. Thus dyspepsia and irregularity of the bowels are common. *Vomiting*, when persistent, is a symptom of considerable gravity, because it is usually of toxic—*i.e.*, uræmic—origin.

§ 294. The Complications and Secondary Inflammations in renal cases are very apt to affect the SEROUS membranes, the MUCOUS membranes, and the SKIN—in a word, the limiting or " surface " structures of the body. The *serous membranes* often become inflamed insidiously, especially the pleura and pericardium. The effusion may sometimes come on very suddenly, but the symptoms may be quite latent ; therefore the occurrence of severe dyspnœa in renal cases should lead us to suspect the sudden supervention of a serious pleural effusion (§ 23). In addition to the *pulmonary œdema* already mentioned, a low form of pneumonia or bronchitis is a common complication of nephritis. Endocarditis is relatively rare. Within the last few years it has been recognised that renal disease may be complicated by various *skin affections* other than dropsy and the cellulitis which is liable to affect dropsical limbs. Amongst these may be mentioned eczema, urticaria, and various forms of erythema. Undoubtedly the most fatal is an epidemic form of exfoliative dermatitis described by the author in 1891.[1] All the cases of renal disease complicated by the epidemic exfoliative dermatitis which the author has since met with, have ended fatally.

§ 295. Pain in the Kidney.—Many serious diseases of the renal substance are unaccompanied by any pain or local symptoms. A sense of dull aching in the loins may be present at the onset of acute nephritis. In pyelitis, lumbar pain generally accompanies the appearance of pus in acid urine. The pain is very severe when the pyuria (pus in the urine) is due to a renal calculus (Renal Colic, § 327). Various tumours of the kidney are accompanied by pain, and perinephric abscesses are associated with lumbar pain and tenderness. A dull, dragging pain or weight in the lumbar region, relieved by rest in the recumbent posture, occurs with movable kidney ; it is usually on the affected side, and is liable to acute exacerbations resembling renal colic. The lumbar pain of renal disease must not be mistaken for the backache due to congestion of the female generative organs, nor for lumbago, in which the pain is usually of sudden onset, is not confined to one side, and may be accompanied by other rheumatic evidences. *Less frequent causes of lumbar pain are reflex pain from conditions of the intestine, aneurysm, cancer, and vertebral caries, etc. (§ 367).*

§ 296. Uræmia is a term used to describe the group of symptoms which arise from retention within the body of those nitrogenous constituents which, under normal circumstances, are elaborated into urea and eliminated by the kidney. The exact nature of these retained substances is not yet known. The term uræmia is generally used for the intense *acutely* toxic

[1] Trans. Med. Soc., London, 1891–1892 ; and *British Journal of Dermatology*, 1892.

condition which closes most renal cases; but it may also be applied to
the incipient or *chronic* condition which precedes this, and warns the
observant physician of the gravity of the situation. It is evidence of
retention in the blood and the tissues of those substances which form a
chain of compounds between the proteid food substances and the nitro-
genous disintegration on the one hand, and the nitrogenous output on
the other. A high urea level in the blood or the spinal fluid is usually
taken as diagnostic of uræmia.

Uræmia, more or less severe, may occur in almost any disease of the
kidney. In renal fibrosis (granular kidney) it occurs in a typically chronic
form; in acute, subacute, and chronic tubal nephritis it is the usual
mode of death; in tuberculous, calculous, and cystic disease, in hydro-
nephrosis and consecutive nephritis, in active or passive congestion, and
in lardaceous disease (rarely), mentioned in order of frequency, it is also
apt to supervene. Moreover, complete suppression of•urine may produce
death associated with symptoms of what is called *latent uræmia* (§ 337),
in those relatively rare cases of blocking of the ureters.

Symptoms.—Various forms (nervous, gastro-intestinal, dyspnœic, etc.)
of uræmia are sometimes described, but it is more convenient and not
more artificial to describe the symptoms under *incipient* and *advanced*
chronic uræmia, and *acute* uræmia.

1. In *incipent chronic* uræmia, such as occurs typically in chronic
interstitial nephritis, the symptoms are vague, and start insidiously. The
patient remains at work, but complains of malaise, headache, loss of
mental and bodily vigour, general wasting of muscular and subcutaneous
tissues, impaired memory, and sometimes sleeplessness after the first few
hours of the night. These and the urinary changes may be the only
indications of the condition.

2. Symptoms of *advanced chronic* uræmia may succeed the foregoing
or may come on abruptly in a person apparently in good health. They
consist of (i.) restlessness, twitching, and muscular tremors (which con-
stitute one of the most constant symptoms); (ii.) persistent headache;
(iii.) drowsiness during the day, with sleeplessness or "cat-sleeps" (dropping
off for a few minutes at a time) at night; (iv.) vomiting, without obvious
dietetic irregularity or gastric disturbance, and sometimes diarrhœa; and
(v.) dyspnœa on slight exertion (which is often the first symptom to be
noticed), or coming on in paroxysms, especially at night. Uræmic
Dyspnœa may be: (i.) *Paroxysmal;* the attacks coming on chiefly at
night, and resembling asthma. The patient sits up in bed gasping for
breath, but there is no cyanosis, and the mind is clear. The breathing
is often noisy, with a characteristic hissing quality (Addison). (ii.) *Con-
tinuous,* or continuous alternating with paroxysmal. (iii.) *Cheyne-Stokes
Respiration* (§ 22), may last for weeks. The pulse slows in the apnœa
period, and there is alternate contraction and dilatation of the pupil, the
contraction occurring during the period of apnœa.

3. *Acute* or *fulminating* uræmia may supervene at any stage of the

foregoing, being ushered in by an increase of the headache, vomiting, or restlessness ; or it may come on abruptly in an apparently healthy person. Its leading symptoms are three : (i.) Low muttering delirium ; (ii.) stupor, passing into coma, with or without (iii.) convulsions. The patient may pass from convulsions to coma, and again to convulsions. In some cases of chronic Bright's disease convulsions or coma may constitute the first manifestation of the disease.[1] Sometimes blindness (uræmic amaurosis), without appreciable ophthalmoscopic changes, follows the convulsions, and may last for several days. Deafness or local paralyses may ensue. There is often a urinous odour in the breath.

Diagnosis.—Uræmia is known by the combination of these symptoms, and the presence of a cause, which can be made out on a careful examination of the urine. In a case of doubtful uræmia or cardio-vascular disease a blood urea estimation usually decides the diagnosis. It must, however, be remembered that the two conditions may exist together. The diagnosis of uræmic coma is dealt with in Chapter XIX, § 564.

The *Treatment* of uræmia is given under Chronic Interstitial Nephritis (§ 323), in which malady both chronic and acute uræmia typically occur.

PART B. PHYSICAL EXAMINATION

The **Examination of the Urine** corresponds, in renal diseases, to the physical examination of other organs.

We examine it by (a) observing its *physical characters* (§ 297)—viz., its appearance (*i.e.*, its colour, and whether it is clear or cloudy)—its odour, reaction, specific gravity ; the presence and characters of any deposit ; and its diurnal quantity. (b) Then by *chemical analysis* (§ 302) we ascertain the presence or absence of albumen, the presence or absence of sugar, and other substances, according to circumstances. (c) A *microscopic examination* (§ 315) has to be made of any deposit which may be present. (d) The *kidney efficiency* tests consist in part of examination of the urine, and in part of examination of the blood. The blood examinations are usually conducted by skilled laboratory workers (§ 311). They are of special value in the detection of nephritis where albuminuria is slight or sometimes absent. It is important in all cases—not only in cases of suspected renal disease—to observe *and to note* the condition of the urine when the patient is first seen, even when the symptoms do not suggest renal disease.

(a) Physical Character of the Urine

§ 297. **Appearance.**—The colour of the urine depends upon the proportion of pigments present. The chief pigments are urobilin and urochrome, whose antecedents are the blood and bile pigments ; but there are many others.

The urine varies from a pale yellow to a deep amber, according to the DEGREE OF DILUTION of the pigments ; and, as the latter are fairly constant in quantity, a *dark urine* is associated with a smaller diurnal quantity and a higher specific gravity than a pale urine. The urine is dark in excessive perspiration, acute nephritis, and pyrexial states generally. On the other hand, in certain diseases with *polyuria* the urine is

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[1] This is explained by a sudden congestion of a chronically diseased kidney, and such cases (coma or convulsions occurring suddenly in an *apparently* healthy person) usually occurred during the winter in the Infirmary.

pale, as in chronic Bright's disease, and in diabetes. In diabetes insipidus, hysteria, and other nervous conditions, the urine may be as colourless as water.

The colour of the urine may be altered by MORBID PRODUCTS—*e.g.*, a *dark orange colour to brown*, having a greenish tint on the surface with reflected light, is due to the presence of bile, and will vary in depth of tint according to the amount of bile present. A *red* colour, which may be a dark red or porter colour or only a more " smokiness," is due to the presence of blood (§ 309). In diseases in which there is destruction of the red blood corpuscles the urine is thickened, and this may be a means of distinguishing pernicious anæmia from chlorosis. *Blackish brown* colour may be due to melanin and certain oxyacids, which cause the urine to darken on exposure. A *bright green* urine may be associated with chloroma. *Milky* urine is found with chyluria and multiple myeloma. Various DRUGS affect the colour of the urine. A *dark olive-green* or *black colour* may be due to the absorption ˌof carbolic acid—as, for example, when this substance is used for dressings ; or it may appear after the administration of creosote, the salicylates, salol, tar, resorcin, or naphthol. The colour is explained by the presence of hydrochinon, which turns crimson on the addition of ferric chloride. A *reddish-brown colour* may be due to rhubarb, senna, or chrysophanic acid when taken internally, and a *bright yellow colour* follows the administration of santonin. All these turn red on the addition of an alkali. A *colourless* urine is said to result from tannin taken by the mouth, and a *reddish* hue from logwood. The urine may be *red* after the application of Scarlet Red ointment to superficial sores. Coloured sweets and cakes may cause a coloured urine, from the presence of eosin, methylene blue, or other dye. *Black* urine may also follow the ingestion of black cherries or bilberries.

Urinary Deposits and Cloudiness will be described in § 314.

§ 298. **Reaction.**—The urine should be tested immediately or soon after being passed. In normal urine an acid reaction is found, turning blue litmus paper red, from the presence of acid phosphate of sodium. On standing for a time decomposition takes place, the urea being transformed into ammonium carbonate $(NH_2)_2CO + 2H_2O = (NH_4)_2CO_3$, and hence the reaction is alkaline. The same change takes place even within the bladder, in cases of chronic catarrh of that organ. Alkalinity occurs even in normal urine after meals—the " alkaline tide "—due to the disodium phosphate $(Na_2HP.4)$ replacing the acid salt, or when a patient is undergoing alkaline treatment. A *neutral* reaction may occur under the same conditions. To distinguish between the alkalinity due to a fixed alkali (*e.g.*, soda or potash salts), and that due to decomposition, ˌwhich depends upon a volatile alkali (ammonia), hold over a flame the red litmus paper which has been turned blue ; if due to a volatile alkali, the red colour will return (as the ammonia is driven off) ; if to a fixed alkali, the blue colour remains.

FIG. 73. — URINOMETER, made of metal, and with flanged foot.— The flange steady it while in the urine, and form a stand when not in use.

§ 299. **Specific Gravity.**—The average specific gravity of the urine varies between 1015 and 1025. It depends chiefly upon two substances normally present : urea and salts (especially chlorides) ; and the simple rule of doubling the last two figures gives roughly a little less than the total quantity of solids present in parts per thousand. Extractives and pigment play only a small part ; and practically—since the salts are fairly constant—the specific gravity, *in the absence of sugar*, gives us a fair measure of the urea present in a given sample. The specific gravity is low in granular, lardaceous, and cystic kidney ; high in acute and sub-acute nephritis and in passive congestion. The specific gravity must be considered in relation to the quantity of urine passed ; and to be able to draw accurate inferences from the specific gravity,

the urine of a whole day should be collected, and a sample thereof tested (§ 301). The instrument used to test the specific gravity is called a urinometer (Fig. 73). The instrument must not touch the sides of the vessel, and the graduated stem should be read along the surface of the fluid, not at the place where it is raised along the stem by capillarity. These instruments are graduated for a temperature of 60° F. If the temperature of the urine is lower than this, the true specific gravity is a trifle lower than the actual reading. It is important to remember that as urine cools, the specific gravity rises. When enough urine is not obtainable, and a glass bead urinometer is not accessible, mix the urine with one, two, or three times its own bulk of water and multiply the last two figures of the specific gravity by two, three, or four, respectively. For example, a mixture of one ounce of urine with *three* ounces of distilled water gives a specific gravity of 1005 ; the specific gravity of the urine was 1020(0·005 × 4 = 0·020).

§ 300. The normal odour of freshly-passed urine is described as " aromatic " ; it is very different from the ammoniacal odour of decomposing urine. The resinous portions of copaiba, cubebs, and other balsams are excreted by the urine, and impart their characteristic odour to it. Turpentine gives to urine an odour said to resemble violets. · It may smell of volatile sulphides due to the presence of some microbes, notably *B. coli communis*, and also where cystinuria is present, especially after the urine has stood for a little.

§ 301. The Diurnal Quantity varies considerably within the range of health. Normally, 40 to 50 ounces (1½ litres) are passed per diem, but the quantity depends upon the amount of fluid drunk, the action of the skin, and the activity of the renal circulation. In order to estimate the quantity of urea, and for some other purposes, it is necessary to collect the whole of the urine that is passed in twenty-four hours—say, for example, from 8 A.M. Monday to 8 A.M. Tuesday. The patient should pass water *at* 8 A.M. *on Monday morning*, and this should be thrown away. Then all that is passed after that hour, together with what is passed *at* 8 A.M. *on Tuesday*, should be collected in one clean vessel, which must be carefully preserved from accident or interference. During the whole of that time it is necessary to pass water *before* going to stool, and to add this to the total collected. At 8 A.M. on Tuesday, after passing water and adding it to that previously passed, the whole should be stirred and measured. A specimen from this should then be put into a clean bottle (say, 10 ounces), and this should be labelled with the name of the patient, the date, and the total quantity passed in twenty-hour hours, and sent for examination immediately.

(b) Chemical Examination of the Urine

Normally the urine consists of water containing about 4 per cent. of solids by weight, of which urea, the most important, comprises from 2·5 to 3 per cent. of the total urine, amounting to about 30 grammes per diem.

In disease the three most important substances for which the urine has to be tested chemically are albumen, sugar, and urea.

§ 302. **Albumen** is the most frequent of the pathological constituents of the urine. The variety of albumen usually present is serum albumen and globulin. Laboratory tests are required to determine the serum globulin ratio. The globulin is increased in functional, reduced in nephritic albuminuria.

The chief tests for albumen are : (1) Cold nitric acid ; (2) Boiling ; (3) Picric acid.

1. *The Cold Nitric Acid Test* is the most delicate, accurate, and convenient test for small quantities of albumen in the urine. Pour some

strong nitric acid into the bottom of the test-tube, hold the tube in a very sloping position, and let the urine gently flow upon the top; a haze of precipitated albumen will appear at the line of junction. It is necessary to wait a few seconds for the haze to appear, when the albumen is very small in quantity; and the tube should be gently heated at the junction.

The *Fallacies* of this test are not serious. (i.) Mucin, or urates, may form a precipitate, but it occurs *above* the line of junction; (ii.) in a concentrated urine, a haze of tiny crystals of nitrate of urea may form, but this may readily be dissolved by heat; (iii.) copaiba and other resins give a haze in a similar position, but the odour is characteristic; (iv.) The haze due to the presence of albumoses disappears on heating, and reappears on cooling; (v.) both pus and blood contain albumen, and if present in the urine, give this reaction, apart from the presence of free albumen. ᶠ

2. *Boiling.*—After testing with litmus, boil the urine, and afterwards add a drop or two of acetic acid. A generalised white precipitate forms on boiling if albumen is present, and is not dissolved by acetic acid. It is always best to boil the upper part of a column of urine so as to compare it with the lower.

Where no test-tube is available at the bedside, it is useful to remember that the urine may be boiled in an iron spoon, and a little vinegar used instead of acetic acid.

The *Fallacies* of this test are: (i.) *Phosphates* may be precipitated by heat alone if the urine be faintly acid, neutral, or alkaline, but the acetic acid dissolves these and increases the albuminous precipitate. (ii.) Excess of acid may redissolve the albumen; undue natural acidity may have the same effect; all of which prove the usefulness of test-papers. (iii.) In acid urines a cloud sometimes appears, not on boiling only, as albumen would do, but when the acid is added, due to mucus. (iv.) Copaiba and other resins may give a precipitate insoluble in acid, but their odour is characteristic. (v.) If the urine is not quite clear, it may be necessary to filter it. If turbid from bacteria, add a trace of NaOH, and a deposit of phosphates occurs which carries the bacteria down with it. Filter and acidify again before testing.

3. *Picric Acid Test.*—Float carefully a saturated solution of picric acid on the urine by a pipette. A precipitate forming at the line of junction of the fluids indicates the presence of albumen. Urates, alkaloids, and albumoses may also be precipitated, but disappear on heating.

The *quantitative estimation* of albumen may be roughly determined by boiling as above and setting aside the test-tube for twenty-four hours, and reading off the proportion. It may be more precisely calculated by means of Esbach's albuminometer, a tube graduated for measuring the percentage of albumen. Urine taken from twenty-four hours' collection is poured into the tube up to the mark U, and the reagent [1] is added up to the mark R. The tube is then set aside for twenty-four hours, and the precipitate falls to the bottom. The level to which this reaches is then noted, and the number on the glass indicates the grammes per litre of albumen present. *Fallacies.* —(1) This method is not reliable if the specific gravity of the urine is over 1010. The urine should be diluted to 1010, and a calculation made afterwards by multiplying the result by the number of times of dilution. (2) If the patient is taking alkaline salts, crystals are liable to appear after adding the reagent, and these must be allowed for in reading off the quantity of albumen. Another method is precipitation by boiling, washing the precipitates and weighing.

§ 303. **Nucleo-proteid** occurs sometimes in febrile disorders and in association with destruction of the kidney cells. It gives most of the tests for albumen, but is pre-

Picric acid, 1 part; citric acid, 2 parts; water, 100 parts.

cipitated on the addition of acetic acid.   From mucin it is distinguished by the fact that it is soluble in a large excess of acetic acid, whilst mucin is not.

§ 304.   Mucin is precipitated, as above mentioned, by most of the same reagents as albumen, but it may be detected by taking a saturated solution of citric acid in a test-tube, and trickling the urine down the sloped side of the tube, when a cloud forms above the junction of the fluids.   Mucin is dissolved in alkaline urine.   Excess of mucus indicates irritation of the bladder or genito-urinary tract, or a vaginal or uterine discharge.

§ 305.   **Sugar (Glucose)** is not a normal constituent of the urine, but it may occur as a permanent or temporary pathological product.   The chief cause of glycosuria (sugar in the urine) is Diabetes Mellitus (§ 335).   The sugar may disappear from the urine in this disease for some days, and reappear again as abundantly as before.   Its causes are referred to in § 334.

TESTS FOR GLUCOSE.—(1) *Trommer's Test* constitutes one of the readiest for discovering sugar.   To an inch of urine in a test-tube add one-eighth its volume of caustic potash and a few drops of a solution of copper sulphate.   On boiling, a red precipitate denotes the presence of glucose.

Benedict's Test.—The reagent consists of copper sulphate 17·3 gm., sod. cit. 173 gm., sod. carb. (anhyd.) 100 gm., aq. dest. ad. 1000 c.c.   Add 8 drops urine to a few c.c. of the reagent, and boil 1 or 2 minutes, allow to cool.   If dextrose is present a red, yellow, or green precipitate forms.   A turbidity due to urates may occur.

(2) *Fehling's Test.*—Fehling's solution consists of an alkaline solution of potassio-tartrate of copper, so prepared that 10 c.c. is reduced by 0·05 gramme of glucose. As it is apt to alter on keeping, it should be boiled before using, to make certain that no precipitate forms before adding the urine.   It is better to keep the copper solution and the alkali solution in separate bottles, mixing them just before using.   Add to it a few drops of urine and boil again : and then continue adding till equal quantities of urine and Fehling are used.   If on further boiling the solution is still clear, no noteworthy quantity of sugar is present.   The Fehling's solution must always be in excess, and the boiling must not be too prolonged.   This test depends upon the fact that glucose has the property of reducing cupric salts when heated in the presence of a free alkali. $CuSO_4$ added to $NaOH$ causes a pale blue precipitate of hydrated cupric oxide.   If a tartrate is present, the cupric hydrate is held in solution (Fehling's solution).   If glucose or some other readily oxidisable substance is added, this blue cupric hydrate on gently heating is reduced, and falls as a *red* or *yellow* precipitate of cuprous hydrate (Cu_2O, H_2O), which on longer boiling becomes red or purple cuprous oxide (Cu_2O).

Fallacies.—(i.) The urine to be tested must be freed from albumen, and (ii.) it must not be ammoniacal.   (iii.) Other reducing agents may occasionally give the reaction. After the administration of chloroform, chloral, morphia, curare, and some other drugs, a reaction is obtained resembling that due to sugar, but is due probably to the presence of glycuronic acid.   Lactose, uric acid, and urates, ammonium chloride, and other ammonium salts, hippuric acid, kreatinine, oxyacids and the products of certain drugs, such as carbolic or benzoic acids, may occasionally be sources of fallacy. To avoid these it is best to control by the Fermentation Test, or to filter a few drachms of the urine through a charcoal filter seven or eight times, by which means all reducing substances other than sugar are removed.

Quantitative Estimation by Fehling's Solution.—The urine should be a sample taken from the total collection of twenty-four hours.   Fill a burette with urine diluted to 1 in 20, and have 10 c.c. Fehling's solution in a porcelain dish, diluted with water. Boil the solution, and while boiling allow drops of urine to mix with it, stirring all the while.   Urine must be run in from the burette till the fluid is colourless ; this is difficult to decide unless the dish be tilted so that it shows against the white background apart from the red precipitate which collects at the bottom.   Read off the amount of urine

required for complete reduction and calculate. Supposing we find that 60 c.c. *diluted* urine from the burette are required to decolorise the 10 c.c. Fehling (representing 0·05 gramme glucose), then $\frac{10}{60} = 3$ c.c. urine contain 0·05 gramme glucose. Then from this, as we know the number of c.c. urine passed by patient in twenty-four hours, it is easy to calculate the percentage of sugar excreted in that time. Carwardine's Saccharimeter (Fig. 74) may be employed in this process if an ordinary burette, as used in the laboratory, is not accessible.

(2a) *The Ammoniated Cupric Solution* (Pavy's Test) is a modification of Fehling's solution. It contains free ammonia which keeps the oxide in solution, which would otherwise be precipitated by the addition of diabetic urine. Hence the *blue colour* of the fluid is discharged *without the formation of any precipitate*, and it is thus easier to determine the exact point when the whole of the cupric salt is reduced than is the case with Fehling's solution. Pavy's solution (10 c.c. of which represent 0·005 gramme of sugar) is therefore very useful as a quantitative test. It is usually applied by means of a special apparatus.

(3) The *Fermentation Test* constitutes the ultimate test in all cases of doubt, since
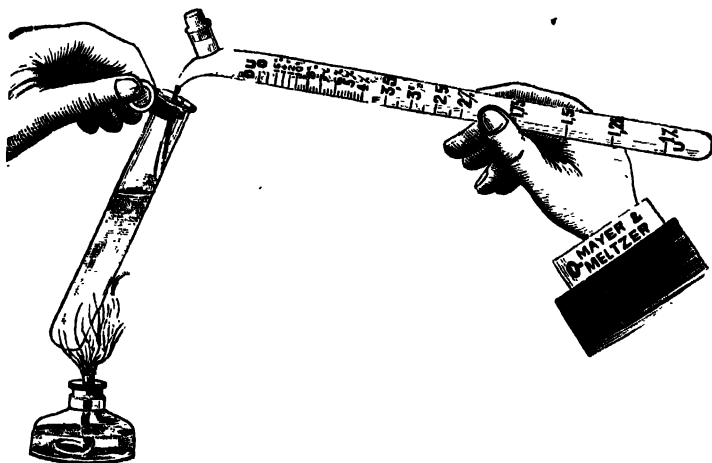
FIG. 74.—CARWARDINE'S SACCHARIMETER.—A sample of the twenty-four hours' collection of urine is used to fill the burette (on right of figure) up to the letter U. Dilute it by adding water to D U, and mix thoroughly. Fill the measure supplied with the apparatus up to F with Fehling's solution, and dilute it by adding water to D F. Pour this diluted Fehling into the test-tube shown in figure and boil it. While it is gently boiling add the diluted urine drop by drop from the burette until all the blue colour has gone from the supernatant fluid. This may take some little time, as it is necessary after each boiling to wait a minute for the precipitate to subside a little. For calculation see text.

sugar is the only known substance fermented by yeast. See that the urine is acid— if need be, acidify with tartaric acid—pour it into a test-tube, and insert a piece of German yeast ; invert the tube over a saucer of water (or mercury) and place in a warm place. Have a control tube beside it with normal urine. If sugar is present, bubbles of CO_2 form and collect at the top of the tube.

This test can also be applied for the *quantitative estimation* of sugar, by Robert's differential density test. *Method.*—Have two 12-ounce bottles with a slit cut in the side of the corks for the gas to escape, and put in each 4 ounces of the urine taken from a twenty-four hours' sample. To one add a piece of German yeast, the size of a walnut, and set them aside in a slightly warm place for 24 hours. Take the specific gravity of both samples, and the difference between them gives the measure of the sugar in grains per ounce. The percentage is found by multiplying this difference by 0·23. Thus, supposing the specific gravity of the two samples is 1050 and 1005

respectively, there were 45 grains of sugar per ounce, and 45 × 0·23 = 10·35 per cent. It is important to wait until all fermentation has ceased, and to see that no decomposition of urea has taken place in the control bottle.

(4) *Picric Acid Test.*—Boil a few drops of liq. potassæ with a saturated solution of picric acid. Add urine and boil; a dark opaque red colour denotes glucose. The picric acid must be tested first; if impure it darkens on heating with liq. potassæ. Normal urine darkens, but not so deeply, and remains transparent.

(5) *Phenyl-Hydrazine Test.*—To ʒi of urine in a test-tube add gr. 4 (0·2) phenyl-hydrazine hydrochloride and gr. 2 (0·12) of sodium acetate; boil in a water-bath for half an hour. Cool by placing the tube in cold water. A yellow deposit forms, which under the microscope shows fine yellow needle-shaped crystals in sheaves.

Lactosuria.—Lactose may be present in the urine in considerable quantity in nursing women. Lactose does not answer to the fermentation test, but it reduces Fehling's solution. In calculating results remember that 10 parts of lactose have the same reducing power as 7 parts of glucose. **Pentosuria** reduces the alkaline copper solution, but does not give the fermentation test. It is a rare error of metabolism.

§ 306. Urea.—A healthy male adult, weighing, say, 140 pounds, excretes about *3·5* grains of urea per pound of his body-weight (0·5 gramme per kilo). We may say, therefore, in round figures, that he excretes daily about *50* ounces of urine, *500* grains urea, and that the urine contains about *2·3* per cent. of urea,[1] the corresponding figures on the metrical system being approximately 1,320 c.c., 33 grammes, and 2·3 grammes per 100 c.c. These figures vary widely in health, and are much less (say 300 grains) for a lighter person taking less food. If the kidneys are acting well, the urea output is increased by an increase in the nitrogenous food. On the other hand, it is considerably diminished after vomiting or diarrhœa. Particulars on all of these points should be noted. A *specimen for estimation should be taken from the urine of twenty-four hours, mixed and measured* (§ 301). Finally, more than one observation should be made before concluding that there is deficient nitrogenous elimination. Deficient elimination of urea occurs sooner or later in nearly all renal diseases (the accompanying effect being urœmia, § 296), in certain hepatic diseases, in myxœdema, Addison's disease, and melancholia. In certain cases the blood urea must also be estimated. (See Kidney Efficiency tests, § 311.)

ESTIMATION OF UREA.—Without an estimation of the nitrogen in the food the measure of the urea excreted is a defective guide. The specific gravity of the urine gives us (in the absence of sugar) a very fair idea of the quantity of urea being excreted; indeed, that is the chief reason why we habitually use the urinometer (§ 299). The rapid crystallisation of nitrate of urea in a test-tube when an equal bulk of strong nitric acid is added to the urine and the mixture cooled, suggests excess; but for accurate results it is necessary to determine the *total nitrogen* in the urine (the greater portion of this being in the form of urea) by volumetric analysis. This has been now rendered available for clinical practice by the simple apparatus described below.

The apparatus usually employed is Gerrard's Ureameter or some modification of it. A wide-mouthed bottle is fitted with a rubber stopper through which passes tubing connected with a graduated glass cylinder. At the upper end of the cylinder is a cork through which runs tubing with a stopcock; from its lower end a rubber

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[1] It is useful to know that the number of *grains per ounce* multiplied by 0·23 gives the *percentage.*

tube passes to an open and short glass vessel which acts as a water reservoir; it can be slipped along the measured cylinder by a metal ring (see Fig. 75). 25 c.c. of freshly prepared solution of sodium hypobromide are placed in the wide-mouthed jar. If the urine contains albumin this must first be removed. Then a small tube containing 5 c.c. of urine is carefully introduced so that it stands up against the side of the wide glass jar, which is tightly stopped. Next, the reservoir is filled with water; the stopcock of the cylinder is opened and the reservoir raised till the water in the cylinder is at zero and level with that in the reservoir. The stopcock is then closed. The next step is to tilt the wide-mouthed jar so that the urine mixes with the hypobromide. Effervescence occurs as the liberated nitrogen enters the cylinder and displaces the water, driving it out into the reservoir. After allowing to cool for ten minutes, the reservoir is moved until the water in it and the cylinder are level; then the amount of gas is read. The cylinder is graduated in percentage of urea, and by multiplying this by 4·375 one obtains the number of grains per ounce.
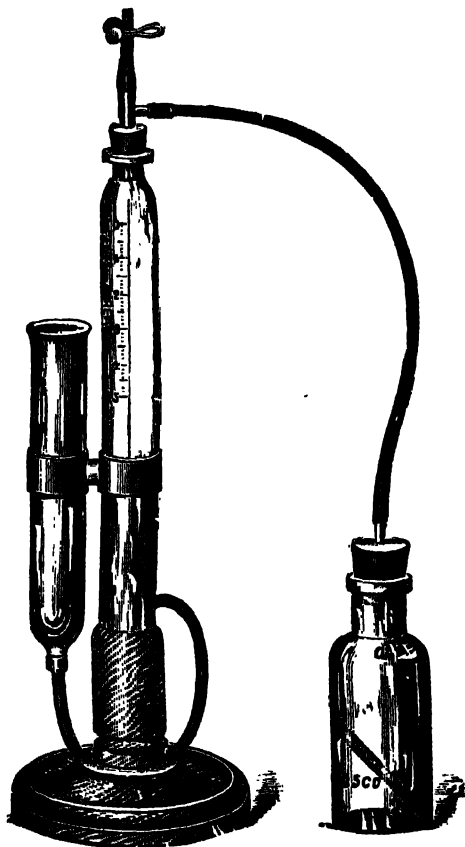
FIG. 75.—GERRARD'S UREAMETER.

§ 307. Uric Acid, either free or combined in the form of urates, is normally present in a sample from a day's collection to the extent of 0·04 per cent., or about 8 or 9 grains per diem. There is in health a fairly constant proportion to the amount of urea, 1 grain of uric acid per day (per 10 pounds body-weight) to 35 grains of urea per day. Uric acid and urates separate out as a cloudiness or deposit (§§ 314 and 315) when there is high acidity. Their c h e m i c a l quantitative estimation is a matter of some delicacy and difficulty.

The murexide chemical *test* for uric acid, free or in combination as urates, is performed by adding nitric acid to the suspected deposit in a porcelain dish, heating to dryness, and placing a drop of ammonia on another part of the dish. Where the two join, a characteristic purple coloration appears. If a drop of caustic potash be placed on another part of the dish, a blue coloration appears at the junction. For accurate estimation of free or combined uric acid, Gowland Hopkin's method is usually employed, or that of Haycraft, both of which are laboratory methods.

§ 308. Bile is present in the urine in cases of jaundice, and can be

detected there even before the skin becomes yellow. Both bile pigments (especially bilirubin) and bile are present, the former more abundantly. An orange-green colour of the urine betrays the presence of bile if in more than slight amount.

(i.) *Gmelin's test* for bile pigments : Run impure nitric acid down a conical glass containing urine. As the bile-pigment oxidises, rings of colour form red, violet, and green at the top ; the green indicates bile. (ii.) *Marechal's test :* Add a few drops of tincture of iodine to the surface of the urine in a test-tube by means of a pipette, and a green reaction is obtained. (iii.) *Hay's test.* Sprinkle flowers of sulphur on the surface of the urine. If bile salts are present the sulphur sinks, instead of floating as on normal urine, because the surface tension of a fluid containing bile salts is lowered.

§ 309. Blood in the urine (Hæmaturia) imparts to the urine a character-istic " smoky " colour, and red blood-cells may be identified under the miscroscope (§ 316). A dark colour of different shades may also be imparted to the urine by Methæmoglobinuria, Hæmatoporphyrinuria, Alcaptonuria, and Carbolic Acid. The most delicate test for hæmoglobin is the spectroscopic test (see Fig. 116).

Chemical Test for Blood.—Add a few drops of freshly-prepared tr. guaiaci to the urine and shake, then add excess of ozonic ether. A blue line appears at the junction of the fluids. The same reaction may be obtained by using filter or blotting-paper. Allow a drop of each of the reagents to fall on the paper beside a drop of the urine, noticing the colour at the junction of the three drops. *Fallacies.*—Saliva gives the same reaction, and so do iodides, in patients taking these salts. Pus gives a blue colour with guaiacum alone. It is very important to have the tincture of guaiacum freshly prepared, and to this end it is best to dissolve a little of the resin in rectified spirit at the time when it is used.

Hæmoglobinuria is always present with hæmaturia, because the corpuscles break up. Its presence *alone* is rare, and can only be proved by examining the centrifugalised deposit of absolutely fresh urine under the microscope and finding *no red cells*, although hæmoglobin is present.

Methæmoglobinuria.—The characteristic smoky colour of the urine in hæmaturia of renal origin depends largely on methæmoglobin, a substance formed from hæmo-globin by the action of acid urine. It is this pigment also which is found in Paroxysmal Hæmoglobinuria. It is recognised by the spectroscope.

Hæmatoporphyrinuria (Iron-free Hæmatin in the Urine).—The urine has a dark cherry-red colour like port wine, but gives no guaiacum reaction. It is found in rare congenital conditions (§ 514), and after excessive drugging with sulphonal, and is an indication for at once stopping the drug and giving alkalies freely. It is known by its spectroscopic bands. If these cannot be detected in the urine, the hæmatoporphyrin should be extracted by shaking with acetic ether or amylic alcohol, after adding a few drops of acetic acid.

§ 310. **Pus** in the urine is best detected by the microscope (§ 316). When in considerable quantity it may be detected chemically by the addition of an equal quantity of liq. potassæ to the deposit. A ropy gelatinous mass is formed, which pours from one test-tube to another like a fluid jelly. This test is only applicable when a fair quantity of pus is present. In small quantities it is best to make a micro-scopic examination of the deposit for pus cells. When pus comes from the *kidney*, the urine is, at any rate when first passed, acid, and the pus is *uniformly disseminated* through the urine, and remains so for some time. When it comes from the *bladder*, the urine is alkaline or neutral, and the pus very rapidly collects into a *creamy layer* at the bottom of the glass.

§ 311. **Kidney Efficiency Tests.**—Certain tests yield invaluable evidence as to the condition of the kidney. These have usurped the position of the old tests for the

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amount of urea, which was formerly regarded as the chief sources of information. Several of these tests are beyond the power of the physician, and should be confided to the skilled laboratory worker. This is true especially of the examination of the blood for protein and non-protein *nitrogen*. *Estimation of Blood urea*.—The laboratory method usually employed depends upon the fact that in the soya bean is an enzyme (urease) which converts urea into ammonium carbonate. In the presence of a strong alkali the ammonia is freed and carried by a current of air which is passed through a solution of acid. The amount of ammonia is measured by the amount of acid which is neutralised, and from this the urea can be calculated. In the usual test each c.c. of acid neutralised is equivalent to 10 mgrams. of urea per 100 c.c. of blood. The same methods can be employed for the estimation of urea in the urine and in the cerebrospinal fluid. It must be remembered that no calculation can be correct without taking into account the nitrogen intake in the food. Normally, the blood has 0·02 to 0·05 per cent. of urea. In renal diseases it rises considerably higher; about 100 mgrams. per 100 c.c. blood may be taken as definitely pathological. A high blood urea may occasionally occur without renal disease ; then the prognosis varies with the cause. In doubtful cases the urea in the urine should be measured ; if this is high, 2·5 to 3·5 per cent., uræmia is excluded if low, about 1 per cent., the excretion of urea by the kidney is defective. The urea concentration test (below) will also decide whether the condition is due to disease of the kidney. As a general rule, there is urea retention in the blood with chronic interstitial nephritis, and not with chronic parenchymatous nephritis. *The Urea concentration test* was introduced by Maclean, and is a very valuable guide to the condition of the kidney substance. The bladder is emptied, and the patient swallows 15 gms. urea dissolved in 100 c.c. water. The urine passed one and two hours later is measured and tested ; the second sample is the more important because diuresis is present at first. If diuresis continues, the urine of the third hour should be examined. About 150 c.c. should be passed per hour ; the urea concentration would be low if as much as 350 to 600 c.c. were passed. Normally 2 to 4 per cent. urea is found. In chronic interstitial nephritis there is less ; serious cases show a concentration of only 1·4 or 1·5 per cent. urea. The *Diastase test* should be carried out at the same time ; when the urea is low the diastase is also low as a rule. Diastase is the ferment which changes starch into sugar. In health, 6 to 30 units of diastase are excreted daily in the urine. When renal activity is impaired, as in nephritis, there is less diastase ; in toxæmias of pregnancy and in mercurial nephritis the index is high ; in early pancreatic disease it is increased up to 300 or 400 units. The test measures the amount of starch digested in a given time by a definite quantity of urine, the starch not changed being indicated by a blue colour on addition of iodine. A 24 hours' sample of urine is required, also (1) 0·1 per cent. solution of soluble starch ; (2) 0·9 per cent. solution of NaCl ; and (3) 1/10 normal solution of iodine. Five test-tubes are numbered 1 to 5. With 1 c.c. pipettes graduated in 1/100ths, place in tube No. 1, 1 c.c. urine ; in No. 2, 0·6 c.c. ; in No. 3, 0·3 c.c. ; in No. 4, 0·2 c.c. ; and in No. 5, 0·1 c.c. of urine. Fill with the saline solution up to 1 c.c. in each tube ; then add to each tube 2 c.c. of the starch solution. Shake and incubate or stand in a hot water-bath at 37° C. for half an hour. Then pour in cold water to within an inch of the top of the tube, so as to arrest the ferment action. Add a drop of the iodine solution to each tube, and shake. Notice which tubes are blue and which are colourless or only a faint pink ; the latter contains the quantity of urine just sufficient to digest the starch. Divide 2 (the number of c.c. of starch) by the number of c.c. of urine in that tube. Suppose it was tube No. 2. Then $\frac{2}{0·6} = 3·3$ units of diastase. Ten tubes may be used, but five are usually enough. When much urine is required, it may be necessary to add two drops of iodine solution. The *phenolsulphonephthalein* test can also be used to detect whether the kidney function is impaired. After the patient has drunk 300 c.c. water, inject intramuscularly 6 milligrammes of the dye, and examine the urine one hour and two hours after. If 40 to 50 per cent. of the dye is excreted in an hour, 70 to 90 per cent. in two hours,

the kidneys are not diseased. *Ammonia* is excreted in daily quantity $\frac{1}{2}$ to 1 gm. The ammonia nitrogen is 2 to 5 per cent. of the total nitrogen excreted. It is increased when acids are ingested or produced in excess in the body ; therefore in all conditions in which acidosis is present.

CHLORIDES.—The chlorides found in the urine are principally salts of sodium, and vary in *health*, according to the food taken, from about 11 to 15 grammes daily. In *disease*, the chlorides are increased during convalescence from fevers, during the stage of absorption of œdema or other forms of serous exudations, and in diabetes insipidus. Except in malaria, they are diminished in acute fevers, especially in pneumonia (reappearing 2 or 3 days after the crisis), in renal diseases with albuminuria, in gastric disease, such as cancer or dilatation, where the digestive power is diminished, in anæmic conditions, and, it is said, in melancholia, idiocy, and dementia.

Test.—Add a few drops of HNO$_3$ to the urine, and an equal bulk of 3 per cent. solution of AgNO$_3$. A curdy precipitate follows if the chlorides are normal in quantity ; if the urine only becomes milky, they are diminished.

Quantitative Estimation of Chlorides (Mohr's Method).—After the urine has been freed from albumen, take 10 c.c. and mix with it 50 c.c. of distilled water ; then add a pinch of calcium carbonate and 3 drops of a neutral chromate of potassium solution (1 in 20). The calcium carbonate neutralises any free acid which may be present. To this a standard solution of silver nitrate is slowly added from a burette, the mixture being stirred constantly. The white precipitate of chloride of silver separates first, but the silver nitrate solution must be added drop by drop until the faintest tinge of pink appears. The pink colour is an indication that chromate of silver is now being formed, all the chlorides having first united with the silver. *Calculation.*—Take the total number of c.c. of silver nitrate used, and deduct 1 c.c. to account for other substances present in urine which unite with the silver ; then every remaining c.c. of the solution used represents 10 milligrammes of sodium chloride.

PHOSPHATES occur in two groups : the alkaline phosphates, salts of potassium, sodium and ammonium ; and the earthy phosphates, salts of calcium and magnesium. The former are readily soluble ; the latter are readily deposited when the urine is not acid, especially when heated.

Tests.—In an *alkaline* or neutral urine, the earthy phosphates form a cloudy precipitate, which is increased on boiling, but disappears on acidifying the urine. If present in an alkaline urine the deposit is distinguished from pus by the fact that it is dissolved by acetic acid. The microscope enables us to distinguish between pus and phosphates, and is indispensable when, as often happens, the two deposits occur together. If the urine be *acid*, it is necessary first to add some caustic potash ; and if it be then heated, the phosphates are precipitated.

SULPHATES are also normally present in the urine, and there is a total increase with increase of diet or fever. They exist in two forms : (*a*) as potassium or sodium sulphate (*inorganic sulphates*) ; (*b*) as combinations of cresol, phenol, indol, skatol, etc. (*organic or ethereal sulphates*). A relative increase of the latter group is of considerable importance. It occurs when phenol or allied substances are given as drugs, and as the result of the action of putrefactive organisms on intestinal contents or abscesses. The exact determination of the total sulphates or of the proportion of inorganic to organic is too complicated for ordinary clinical work. To gauge roughly the proportion of organic sulphates, add to the urine an equal volume of alkaline barium chloride solution. This precipitates the inorganic sulphates and phosphates. Filter : add hydrochloric acid until the filtrate is strongly acid, and heat. The organic sulphates are thus rendered inorganic, and are precipitated. They should normally form a white cloud, and if the precipitate is dense the proportion of organic sulphates is abnormally high.

§ 812. **Proteins in the Urine.**—Besides serum albumen, hæmoglobin, methæmoglobin, hæmatoporphyrin, and mucin and nucleo-albumen the only proteid which in the present state of our knowledge has any clinical significance is albumose. For further details the reader is referred to larger works on the subject.

Albumosuria was formerly known as **Peptonuria**, but it is now supposed that true

peptones never appear in the urine. Albumosuria occurs where there is great destruction of white corpuscles, and therefore whenever there is a large collection of pus in the body—*e.g.*, in empyema and any abscess formation. It is useful in deciding the character of an effusion, pleural, peritoneal, or meningeal. Albumosuria also appears in the resolution stage of pneumonia, and has been described in connection with certain liver diseases, such as acute yellow atrophy, with ulceration of the intestine, with dyspepsia, sometimes when excess of animal food is consumed, and with some cases of nephritis (together, of course, with albuminuria). It may be the first sign of that rare disease, myelopathic albumosuria or Kahler's disease (§ 480).

Test.—Primary and secondary albumoses are found, the latter being more nearly related to the peptones—but they have the same clinical significance. (1) Presuming the urine to be free from ordinary albumen, add HNO_3 drop by drop to the urine ; if a precipitate is formed, which disappears on heating and reappears on cooling, *primary albumoses* (? peptones) are present. Both forms of albumose react to the next test. (2) Acidify the urine strongly with acetic acid, add an equal bulk of saturated salt solution till a cloud forms ; if it disappears on heating and reappears on cooling it is due to albumose.

§ 313. **Other Constituents** sometimes met with in the urine are aceto-acetic acid, often called diacetic acid, acetone, dioxyphenylacetic acid, indican and skatol.

Diacetic acid appears when ketosis, described below, is present. It becomes oxidised in the bladder into acetone. Its presence is detected by adding a few drops of a strong aqueous solution of ferric chloride, when a Burgundy-red colour appears.

ACETONE is found in the condition known as **ketosis**, formerly called acidosis. Diacetic acid and acetone have long been known to be present in the urine in cases of Diabetes Mellitus. They were formerly thought to be of grave import, but recent investigation has shown that they may arise from a variety of causes. The diacetic radicle is the important factor, not because it is acid, but because it contains a toxic group. Acidosis is due to a decrease in the fixed bases of the blood and tissues ; though the blood remains alkaline the balance of acids and bases is disturbed. The normal ratio is upheld by the elimination of CO_2 by the lungs and of acid by the kidneys, by the neutralisation of acid by ammonia, and by the intake of bases with food. The acid-base ratio can be upset by derangement of the protein and fat metabolism, as with impaired hepatic function, by kidney disease interfering with acid excretion, and by lack of proper food. In acidosis there is deficiency of utilisable carbohydrate and the subsequent incomplete oxidation of fats leads to the formation of oxybutyric and diacetic acid. The tissues are depleted of bases which go to neutralise the acidity. Much of the nitrogen excreted appears in the urine as salts of ammonia instead of the normal urea. Hence the ammonia coefficient is increased from the normal 5 to as much as 30 to 50 per cent.

The clinical *symptoms* of ketosis are dyspnœa (air-hunger) without cyanosis, and an ethereal odour of the breath. It can be diagnosed by the odour of the breath and by the presence of the ketone substances in the urine as shown by the test described below. Chemical tests reveal the presence, but not the degree of the ketosis. Whereas the normal urine is rendered alkaline after a dose of about a dram of the drug, in ketosis the urine does not become alkaline till about eight times as much has been taken. The clinical *causes* are diabetes mellitus, starvation and inanition, prolonged vomiting and gastro-intestinal diseases which prevent assimilation, certain kidney conditions, diseases of the liver, such as acute yellow atrophy, delayed chloroform poisoning, eclampsia, and febrile states. The cyclical vomiting of children (§ 215) is associated with ketosis due to defective protein and fat metabolism arising from an unknown cause. When the attack is over, a search should be made for septic foci (tonsils, appendix, etc.), as possible sources of toxæmia and hence of the ketosis.

Treatment.—Formerly bicarbonate of soda was given, with the idea of neutralising the excess of fatty acids. Now it is realised that the toxic element is the diacetic radicle. Water aids its elimination. When the ketosis occurs with diabetes mellitus,

see § 335.   Give plenty of water and alkalies till the urine is alkaline, and 1 pint of a 5 per cent. solution of glucose per rectum every four hours.

Test.—Freshly dissolve a crystal of sodium nitroprusside in water and add a few drops of this to 5 c.c. of urine in a test tube ; then pour liq. ammon. fort. on the top ; a purple colour appears at the line of junction and spreads upwards.   The sensitiveness of the reaction can be greatly increased by previous saturation of the urine with crystals of ammonium sulphate.

Alcaptonuria is a condition where the urine forms a pellicle on the surface and darkens from the surface downwards on standing exposed to the air, due to the presence of dioxyphenyl acetic acid.   It is an inborn error of metabolism, and has no known clinical significance.   Its only importance lies in the fact that it reduces Fehling's solution and leads to difficulty in life insurance.

Indicaguria.—Indican is found where there is undue intestinal putrefaction ; usually where bacterial infection is present above the cæcum.

Test.—Add 5 c.c. strong fuming HCl and one drop of a 1 per cent. solution of potassium chlorate to 5 c.c. of urine.   This, on standing for a few minutes, produces a blue colour due to indican, which may be extracted by shaking up with about one-third its volume of chloroform.   *Fallacy.*—A pink colour develops in the urine of patients taking iodides, and when skatol is present.

(c) *The Urinary Deposit.*

§ 314. **Cloudiness of the Urine** (naked-eye examination).   In healthy urine there is no deposit, but most of the normal constituents, and some abnormal substances, may become evident as a sediment or turbidity after the urine has cooled.

(1) A bulky pinkish turbidity and deposit in an acid urine, which forms when the urine cools, indicates the presence of urates.   It is the commonest of urinary deposits, and its appearance when the urine gets cold is typical.   (2) *Uric Acid* is evident to the naked eye as a sandy deposit resembling red cayenne pepper.   (3) A white flocculent turbidity in an alkaline or neutral urine indicates the presence of *phosphates*, which are cleared at once by the addition of a few drops of acetic acid.   (4) *Calcium oxalate* gives a typical " powdered-wig " deposit of fine white points seen on the surface of a mucous cloud.   (5) A fine cloud of *vesical mucus* is normally present in the urine, although it is only visible when the entangled débris and epithelial cells are sufficiently plentiful.   (6) *Pus* forms a deposit which resembles phosphates to the naked eye, but it is readily distinguished under the microscope.   (7) Urine is sometimes cloudy from the presence of *bacteria*, and this cloudiness cannot be cleared by boiling or the addition of acids.

§ 315. **Specimens** of the deposit must always be examined microscopically in cases of suspected renal disease.   The urinary deposit is best examined after the urine has stood for some hours in a conical glass, or after the specimen has been centrifugalised.[1] Take a pipette, close it at the top with the right forefinger, pass it to the bottom of the glass, allow a small quantity of the sediment to enter, withdraw the pipette, wipe its exterior with a cloth, place the point on a slide, then surround the pipette with the palm of the left hand, the warmth of which will cause a drop to exude.   Cover the drop with a cover-glass, and examine first under a ¼ or ½ inch objective, then

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[1] For centrifugalisation a special apparatus is necessary, the specimen being placed in a tube on the edge of a rapidly rotating wheel.   It is convenient, and saves time.

under a ⅓ or higher.   The deposit normally contains foreign substances, such as cotton and woollen fibres, etc., and a few bladder (and in women nearly always a few vaginal) epithelial cells, which are recognised by their large and nucleated appearance.   Inquiry should always be made as to the sex of the patient, and in women if any leucorrhœa is present.   If so, it is very desirable to draw off a specimen of urine by the catheter.

The urinary deposit may contain ORGANISED SUBSTANCES (§ 316), or CRYSTALLINE or unorganised substances (§ 317).

§ 316. The **Organised Constituents** of the urinary sediment are of far more serious import than the crystalline substances.   They comprise TUBE-CASTS (which are the most important), EPITHELIAL CELLS, PUS CELLS,
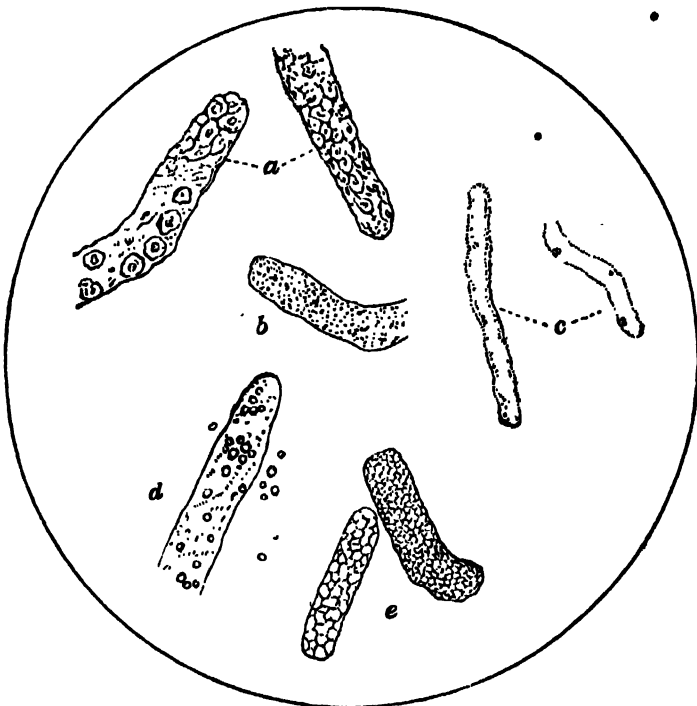
FIG. 76.-—RENAL TUBE-CASTS.—*a*, epithelial casts ; *b*, granular cast ; *c*, hyaline casts ; *d*, fatty cast ;
e, blood casts.

BLOOD CELLS, spermatozoa, and certain rarer structures such as microbes, fat cells, etc.

Tube-casts and renal **Epithelial Cells** are present in all renal maladies attended by shedding or destruction of the renal epithelium.   When tube-casts are abundant in the urine microscopic examination of the sediment permits of their ready detection.   But if, on the other hand, they are present only in small numbers, they may be easily overlooked, and this all the more so when, as in chronic interstitial nephritis and in amyloid disease, the urine is abundant and of low specific gravity, so that any suspended matter it contains is deposited only slowly and incompletely.

Moreover, these are the exact instances in which the casts are apt to be of the hyaline variety, and their almost transparent character renders them inconspicuous objects in the microscopic field.   Hence the search for tube-casts must be conducted with great care if the risk of a false conclusion is to be avoided.   One of the best methods, after settlement or centrifugalisation of the deposit, is to examine it with a moderately low power of the microscope, using a narrow diaphragm and shading the light so as to have the field only feebly illuminated.   Any. suspicious-looking object can be brought into the centre of the field and examined with a stronger lens.   In this way casts may be detected which in a strong light would readily be missed, and if several slides have been prepared and examined in this manner the detection of any casts present in the urine is rendered fairly certain.   But the examination should be repeated on several occasions in any urine containing albumen before a negative conclusion is finally arrived at.   The addition of a few drops of methylene blue to the urine before centrifugalisation is of assistance.   The casts do not stain at first, but in those containing cells the nuclei stain ; and the casts stand out against the pale blue background of the fluid.   There is often a special degree of difficulty in finding casts in alkaline urine, and in decomposing urine they undergo disintegration.

The clinical importance of tube-casts in the urine is that, with but few exceptions, they definitely indicate disease of the renal epithelium.   Thus, when found in a urine containing albumen, they add great weight to the opinion that the albuminuria is a result of some structural change of the kidney.   Similarly in cases of pyuria and hæmaturia the detection of tube-casts not only suggests that the pus and blood are of renal origin, but also that the kidney is becoming affected.   It must be remembered that more than one part of the urinary tract may be diseased at one and the same time.   In the urine of patients who are jaundiced, tube-casts may often be found without, either at the time or subsequently, any evidence of renal disease.

In general terms, *epithelial casts* and *blood casts* are indicative of the earlier and more acute stages of parenchymatous nephritis.   *Waxy casts* are not peculiar to lardaceous kidney, but occur in other forms of long standing renal disease.   These and *fatty casts* indicate that the inflammatory process is passing to a degenerative stage. *Granular casts* are more abundant in chronic renal disease, both tubal and interstitial. *Hyaline casts,* which must not be confused with waxy casts, occur in all forms of nephritis, both acute and chronic, and also in health after middle age.   The relative proportion of epithelial, granular, and hyaline casts (Fig. 76) is affected by the condition of the urine.   In highly acid and in alkaline urine the casts tend to be hyaline ; in acid urine, granular.   Tube-casts in abundance always form a serious symptom, but one or two casts may occur in normal urine.   They are more abundant in the acute than the chronic forms of renal disease.   Their *absence* does not count for very much, as they may be easily missed or undergo disintegration in the urine.   The continued presence of hyaline and granular casts is more serious than the temporary appearance of other types.

Renal Epithelium (Fig. 77).—The detection of renal epithelium in a

urinary deposit has much the same significance as the presence of tube-casts. The cells are *spherical* and rather smaller than bladder or vaginal epithelium. They may be seen isolated or in small groups. In acute Bright's disease they may be found in an unaltered condition, but in chronic disease they become degenerated, and may thus appear crowded with fat globules. BLADDER or VAGINAL EPITHELIUM (Fig. 78) is met with as collections of squamous cells; transitional, spindle-shaped, and other forms of epithelium may also be derived from the bladder. TAILED EPITHELIUM may be derived from the pelvis of the kidney, and the presence of cells having this elongated character would greatly aid the diagnosis in a case of suspected pyelitis. It must be remembered, however, that the male urethra and the prostate gland yield epithelium practically identical with the above. A deposit from this source is not uncommon in cases of chronic prostatitis, the result of a former gonorrhœa.

FIG. 77.—RENAL EPITHELIUM—*a*, normal; *b*, fatty; *c*, disintegrating.
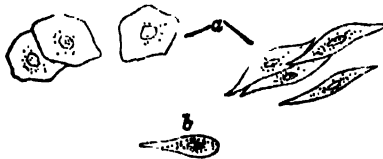
FIG. 78.—BLADDER EPITHELIAL CELLS (*a*); and TAILED EPITHELIUM (*b*) from the pelvis of the kidney.

Pus Corpuscles, under the microscope, are of globular form with a diameter about one-third larger than that of a red blood-cell. The corpuscles are opaque and granular, but when treated with acetic acid they clear up, and a nucleus is seen (Fig. 79, *a* and *b*). Pus cor-puscles may or may not accom-pany bacilli in the urine.

Red Blood Corpuscles.—The detection of red blood-corpuscles in a urinary deposit is, of course, conclusive evidence of the presence of blood. In most fresh urines they are readily distinguished, as they retain their bi-concave form and the outline shows a double contour (Fig. 79, *c*). But sometimes the corpuscles become much changed. Thus in a very dilute urine they are apt to become distended by imbibition, and then are seen as circles having sharp delicate outlines (*d*). In other instances they become crenate, shrunken, and deformed (*e* and *f*).

Spermatozoa may occasionally be found in the urine. Each has a minute oval or pear-shaped head, from the larger extremity of which there passes a long and delicate tail. The total measurement of the spermatozoon is about $\frac{1}{500}$ inch in length.

Micro-organisms.—Numerous microbes are found in the urine, especially when decomposition has occurred within the bladder. The most constant are the *Bacillus*

ureæ, Vibriones, and the *Hay bacillus* (bacillus subtilis), which have no special clinical significance apart from putrefactive changes. *Gonococcus* is found in cases of gonorrhœa. Streptococci are frequently found·in arthritic cases. The *B. Coli Communis* is sometimes found in the urine in pure culture, (§ 331); it is the most common cause of bacilluria. The *typhoid bacillus* may be abundant in cases of enteric fever, and long after health is restored it may remain a potent source of infection to others.

The *Tubercle Bacillus* may be found in tuberculous disease of the bladder or pelvis of the kidney, and is therefore a sign of great value. In appearance under the microscope it resembles the smegma bacillus. Its special staining reaction is given in § 667. It is difficult to find in the urine early in the disease, and in obscure cases
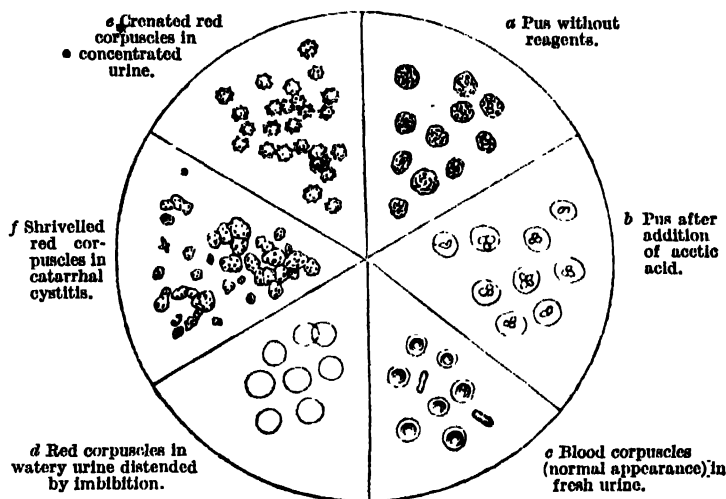
FIG. 79.—Various appearances of RED BLOOD CORPUSCLES and PUS CELLS.—In very pale, watery urine the red corpuscles may be so pale as to escape detection (*d*). They may then be revealed by adding a solution of iodine in potassium iodide.

the experimental test upon guinea-pigs should be employed, the urine for inoculation being collected through a sterilised catheter into a sterilised bottle.

§ 317. Crystalline and Inorganic Deposits in a urinary deposit are usually of less serious import than the organised substances above noted.

In ACID URINES we meet chiefly with urates, uric acid, oxalates, and —among the rarer substances—stellar phosphates, cystin, xanthin, hippuric acid, tyrosin, and leucin.

In neutral or ALKALINE URINES we meet chiefly with triple phosphates (occasionally urate of ammonium and calcium carbonate).

Amorphous deposits of urate of potash or ammonia, and phosphates and carbonates of the alkaline earths may be met with in urines of EITHER REACTION.

1. URATES, chiefly of sodium, potassium, or ammonium, when in excess are deposited as an amorphous brick-coloured deposit after the urine (warm when first passed) has become cold. A deposit having these characters, and disappearing when heated in a test-tube, is sufficiently characteristic for the detection of urates. The deposit is dissolved on the addition of caustic potash ; a test which also distinguishes

urates from phosphates. *Urates of Soda and Potash,* under the microscope, appear as amorphous orange or *pale brown* granules (Fig. 80, *a*). *Urate of Soda* may occasion-
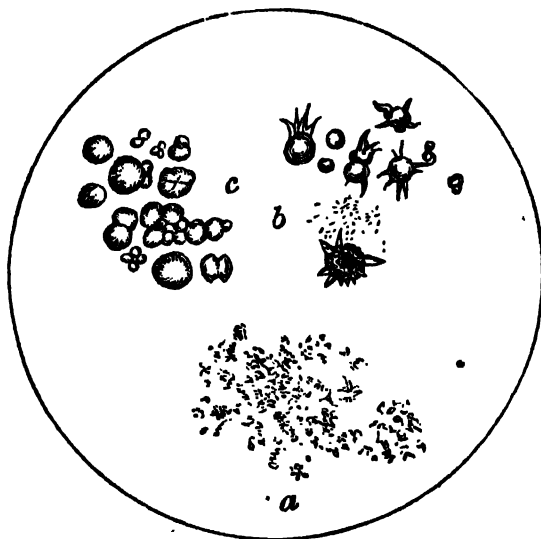

FIG. 80.—URATES.—*a,* Amorphous urates of sodium and potassium ; *b,* "Hedgehog" crystals of sodium urate ; *c,* Ammonium urate.

ally appear as " hedgehog," crystals, globular masses covered with spikes (Fig. 80, *b*) *Urate of Ammonium* occurs as globular masses, sometimes spiked, very like sodium
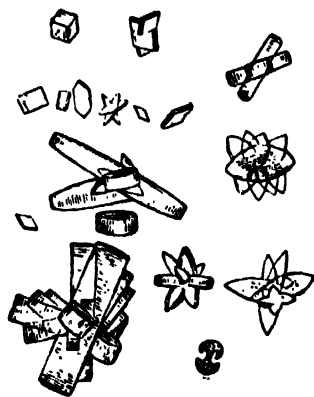

FIG. 81.—URIC ACID crystals (red-brown).—The two top rows show, from left to right, the evolution in a colloid medium of the "lozenge-shaped" crystal from the primary rhombic prism. In the lower right-hand corner is the "dumb-bell" form occasionally met with.

urate, but known from such by being found in alkaline urines (together with phosphates) and by being dissolved by acids (Fig. 80, *c*).

Clinically, urates and uric acid are important only when they occur *constantly,* in fresh urine, or in urine that has stood a few hours only. Gouty and other symptoms are apt to arise in such cases (§ 274), and calculus might be expected to form in the bladder or kidney. An occasional deposit of urates, or a deposit occurring in urine that has stood over six hours, is of but little importance. In all concentrated urines, on cooling, large deposits of urates normally occur. The patient may think the deposit is due to blood.

2. FREE URIC ACID is deposited when the urine is very acid or poor in salts and in pigment, and is therefore found chiefly in dilute pale urines with deficiency of salts. The red deposit of uric acid closely resembles cayenne pepper to the naked eye. It may be detected in the urinary deposit under the microscope by the *colour* and shape of the crystals. It occurs in the form of *red-brown crystals* (the only coloured crystals commonly found in the urine) (Fig. 81). Uric acid assumes many different shapes, owing to the presence of the colloid substances in the urine, but they are all derivatives

from the rhombic prism or parallelogram, in which form uric acid crystallises from pure water. The more pigment, mucus, and other colloids there are in the urine the more spherical do the crystals become. Some of these are shown in the accompanying illustration, and the gradual transition from rhombic prism to dumb-bell and other spherical forms will be seen by following the crystals from left to right. This deposit is soluble in caustic potash, insoluble in dilute acetic acid, the converse of phosphates.

In health uric acid is increased with a highly nitrogenous diet, after much exercise, after meals, and during the " alkaline tide " of the morning. It is also increased after any excess of purin intake, in most fevers, in liver diseases, with deficient intake or excessive loss of fluid, when any cause of leucocytosis is in operation, · during and after acute gout and acute rheumatism. It is diminished in chronic gout, especially just before the acute exacerbations ; in chronic Bright's disease ; in chlorosis and other chronic diseases.

3. PHOSPHATES occur as a white deposit or flocculent turbidity in feebly acid,
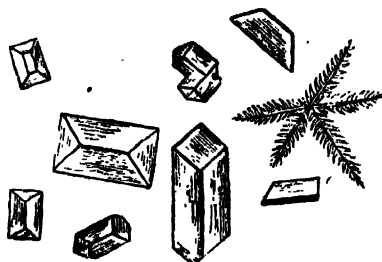
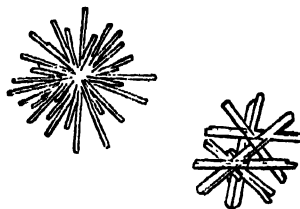FIG. 82.—TRIPLE PHOSPHATE—" house-top ' and " feathery " crystals.

FIG. 84.—NEUTRAL OR " STELLAR " PHOSPHATE.

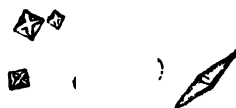FIG. 83.—BASIC MAGNESIUM PHOSPHATE.

FIG. 85.—CALCIUM OXALATE— " envelope " and " dumb-bell " crystals.

NEUTRAL, or ALKALINE urine, in three different forms, which in order of frequency are : (1) *Amorphous phosphates of calcium* form the thick white deposit that is apt to be mistaken for pus, but which is more readily shaken up in the urine. These and all other phosphates are soluble in acetic acid (which distinguishes it from pus). (2) *Triple phosphate* of ammonium and magnesium (Fig. 82) is found in urine which has under-gone alkaline fermentation. The crystals are large colourless three-sided prisms like " house-tops," occurring singly, or as snow-flakes or other irregular forms. In markedly ammoniacal urine " feathery phosphates " are found. (3) *Basic magnesium phosphate* occurs in large rhombic plates, not grouped, but scattered (Fig. 83). (4) *Neutral or dicalcium phosphate* occurs in neutral or alkaline urine as clear, refractile, pointed or wedge-shaped prisms arranged in stellate groups—" stellar phosphates " (Fig. 84). They decompose on the addition of ammonia. The constant presence of phosphatic deposits may be associated with symptoms (§ 340), and usually does not indicate excess eliminated, but only alkalinity of the urine. *Monocalcium phosphate* occurs chiefly in acid urines.

4. OXALATES are chiefly met with as *oxalate of calcium*. This occurs as a scanty crystalline deposit of colourless transparent octohedra, appearing under the micro-scope, like tiny envelopes, hence the name " envelope crystals " (Fig. 85). They

sometimes rest like fine powder above a cloud of mucus, and have been described therefore as the " powdered wig " deposit. They are soluble in hydrochloric acid, insoluble in acetic acid or caustic potash. Oxalate of calcium may also occasionally appear as dumb-bell shaped crystals. These crystals are much clearer and more highly refractile than any other, and atypical forms may be recognised by this feature. The presence of crystals of oxalate of calcium is not necessarily indicative of an excess (OXALURIA, § 340); their presence may also suggest the nature of a calculus. They are said to be abundant in the early stage of chronic pancreatitis.

5. *Calcium Carbonate* is a rare deposit, consisting of tiny spheres and dumb-bells, or of amorphous granules, effervescing and dissolving in acetic acid (Fig. 86). The *Carbonates of the Alkaline Earths* are very occasionally found as tiny amorphous granules or concretions. Calcium sulphate and carbonate may take part in the formation of vesical calculi, especially in the aged, but otherwise they are of no clinical significance. Their presence only points to the existence of a calculus, and indicates its composition.

FIG. 86.—CALCIUM CARBONATE.

When a patient is taking crystalline drugs, such as acetate of potash and phosphate of soda, or even liquor ammoniæ, various crystals which have no pathological significance sometimes appear in the urine. Moreover, after a reagent has been added to urine (*e.g.*, Esbach's solution for the estimation of albumen), and it has been set aside, crystals may also appear which have no clinical value.

6. *Certain rare and less important deposits*, which occur chiefly in acid urines, are as follows : *Hippuric Acid* is an antecedent of uric acid in the nitrogenous metamorphoses of the tissues. It occurs as four-sided prisms, either scattered or in groups. It is present after the ingestion of benzoic acid in large doses, cranberries, and other fruits. *Calcium Sulphate* occurs either as amorphous granules, or, very rarely, as long colourless needles or elongated tables with truncated ends. It is detected by being insoluble in ammonia and acids. *Leucin* occurs as laminated spheroids, and *Tyrosin* as bundles of acicular crystals (Fig. 70). Both occur occasionally in the urine in phosphorous poisoning, acute yellow atrophy of the liver and other causes of liver destruction. *Cholesterin* (Fig. 69) is only occasionally found among urinary deposits. It forms laminated plates with longitudinal striæ, and a notch at one end. *Cystin* occurs as hexagonal plates soluble in ammonia (Fig. 70, § 266).

PHYSICAL EXAMINATION OF THE KIDNEYS

§ 318. A dull " sickening " pain is usually felt on firmly compressing the kidney with both hands, but there is no tenderness in a healthy organ. Tenderness may be elicited in cases of calculus and other forms of pyelitis, perinephric inflammation, abscess, or tumour of the organ. Kidney tumours tend to grow forwards, where there is least resistance, pushing the resonant colon *in front* of them. When, therefore, the palpating hand encounters resistance and swelling in the lumbar region *posteriorly*, it is probably due to a peri- or extra-renal, rather than to a renal condition (see Fig. 36, § 87). The diagnosis of renal swellings from other abdominal tumours has been given in § 212. An extra-renal tumour may press the kidney backwards, so that the apex of the tumour may be due to the displaced kidney.

In the majority of renal disorders the physical examination of the kidney is of secondary importance to the examination of the urine. The kidneys are situated on either side of the spine, about 3 inches from the middle line ; the right is slightly lower

than the left, owing to the position of the liver just above it. The upper end of the right kidney reaches to the *lower* edge of the eleventh rib ; the left kidney reaches as high as the *upper* edge of the eleventh rib. The kidneys lie partly in the hypochondriac and partly in the lumbar regions, and are therefore much higher than is commonly supposed, with reference to the anterior abdominal wall. The lower end of the right kidney is 1 inch and that of the left kidney 1½ inches *above* the level of the umbilicus.

Palpation.—Even in normal conditions the lower border of the right kidney may be palpable in thin people. In those whose abdominal walls are lax—in women who have borne children, for instance—it is surprising how frequently the right kidney can be palpated. The patient should lie on the back, with the abdominal muscles relaxed. The physician, standing on the right of the patient, should place his left hand beneath the patient's back, close under the ribs, just external to the quadratus lumborum. The right hand is laid flat over the anterior surface of the abdomen, in the mid-clavicular line, with the fingers pointing upwards, just below the liver. Pressure · backwards, as if to meet the left hand, is made by the right hand. The patient should then be asked to draw a deep breath, and as he does so the rounded lower edge of the kidney is felt to slip between the opposing hands. When the ligaments of the kidney are relaxed—*movable kidney*—the fingers of the right hand may be able to palpate the upper border of the organ, and to retain it during expiration. A kidney is said to be "*floating*" when it can not only be readily palpated, but can be pushed below the umbilicus or freely moved about in the abdominal cavity.

Percussion does not enable us to define the margins of the kidney, for the organ is too deeply seated. The feature of primary importance in this connection is its relation to the colon, which, as just mentioned, is pushed forward by enlargements or tumours of the kidney. Consequently the anterior surface of such growths is always resonant, there being dulness at the side which is continuous with that at the back ; whereas with enlargements of the spleen or gall-bladder there is dulness anteriorly and resonance at the side.

Other methods of examination of the kidneys are now open to us. In cases of doubtful renal calculus a **radiogram** will usually settle the diagnosis. Examination of the ureteral orifices by means of a **cystoscope** may demonstrate which kidney is affected, for the normal flow of urine may be absent or visibly altered, and the orifice itself may be the seat of infiltration or ulceration. The previous administration of methylene blue or other harmless pigment may make the differences of the flow from the orifices more obvious (**chromo-cystoscopy**). The ureters may be **catheterised**, and a specimen of urine obtained in this way from each kidney. The condition of the bladder is also revealed by the cystoscope. Lastly, **pyelography** has proved useful to determine the condition of the ureters and pelvis of the kidney. A 10 per cent. solution of collargol is injected through a ureteral catheter, and on X-ray examination an opaque shadow is thrown where the solution has penetrated.

PART C. URINARY DISORDERS, THEIR DIAGNOSIS, PROGNOSIS, AND TREATMENT

§ 319. Routine Procedure and Classification.—*First*, having ascertained that the patient's LEADING SYMPTOM refers to the urinary apparatus ; and, *secondly*, the data of his ILLNESS, particularly as to whether it is of an ACUTE or CHRONIC nature ; we proceed, *thirdly*, to examine the urine. The ROUTINE EXAMINATION of the URINE in everyday practice consists of Inspection, Reaction, Specific Gravity, Tests for Albumen and for Sugar. The subsequent and more detailed examination depends upon circumstances. As above stated, the examination of the urine stands in relation to renal disease, as the local signs do to diseases of other organs. There

are very few diseases, certainly no common disorders of the kidneys, which are not attended by some change in the urine. On the other hand, the LOCAL EXAMINATION of the kidney, by palpation and percussion (§ 318), is difficult and relatively much less certain and instructive. On this account it comes last in our scheme of examination, but it should never be omitted in any case which is at all obscure.

Classification.—We will deal with urinary disorders under their respective cardinal symptoms as follows :

Albuminuria	§ 320
Hæmaturia	§ 326
Pyuria	§ 329
Alterations in the specific gravity	§ 332
Polyuria	§ 333
Glycosuria	§ 334
Retention of urine	§ 337
Suppression of urine	§ 338
Incontinence of urine	§ 339
Presence of various deposits	§ 340
Renal enlargements	§ 341

§ 320. Albuminuria.—The numerous morbid conditions which may give rise to albuminuria may be divided into three great anatomical (and clinical) groups : A. *Acute Inflammation* of the Epithelium (Acute Nephritis or Acute Bright's Disease) ; B. *Chronic Inflammations* and *Degenerations ;* C. *Renal Congestions,* either active or passive, which include many cases of albuminuria, independent of structural disease of the kidney.

If, therefore, the illness came on recently, and is of an **Acute** character, turn first to § 321 (Acute Nephritis), and then to § 325 (Renal Congestions).

If, on the other hand, the illness is of some duration, and evidently of a **Chronic** kind, turn first to § 322 (Chronic Tubal Nephritis), and then to the succeeding sections.

When the albumen is in small quantity, and there is also BLOOD or PUS in the urine, turn to § 326 (Hæmaturia), or § 329 (Pyuria), respectively.

The illness came on recently, and is **acute** *; the urine is diminished,* and *contains a considerable quantity of* ALBUMEN *and* TUBE-CASTS *; it is or has been "* SMOKY *" from the presence of blood ;* ANASARCA *is present ; and there is a tendency to uræmia.* The disease is ACUTE NEPHRITIS.

§ 321. Acute Nephritis (Acute Bright's Disease).—In this disease the inflammation begins and predominates in the epithelium or parenchyma of the organ. The condition usually lasts from five to six weeks, and may terminate in recovery or pass on to a chronic condition.

Symptoms.—(1) The albumen is often in considerable quantity, and the urine may even " go solid " on boiling. (2) The other characters of the urine are : (i.) It is scanty, sometimes only 10 or 20 ounces a day, or less. Consequently, the specific gravity is high, although the diurnal quantity of urea is diminished. (ii.) It varies from a turbid or " smoky " to a dark brown hue from the presence of blood. (iii.) Epithelial, hyaline, and blood casts, free renal epithelium, and red blood-corpuscles are present.

(3) Dropsy is general from the commencement, although it is first noticed in the face in the loose areolar tissue below the eyes and in the genitals. There may also be collections of dropsical fluid in the serous cavities. (4) There is a waxy pallor of the skin. (5) A degree of malaise, with discomfort and even pain in the loins, may be present, but there is only a slight elevation of temperature for about four or five days. (6) Uræmic symptoms may come on early—e.g., (i.) occasional vomiting, (ii.) headache, (iii.) drowsiness. (7) In the course of a few days the blood-pressure may become high, and the second aortic sound accentuated.

Causes.—Acute nephritis is only rarely a primary malady. (1) A large percentage of the cases supervene on an acute specific fever, and by far the most common of these is scarlet fever. (2) Septic infection from the tonsils, from wounds, malignant endocarditis and other sources of septicæmia. (3) Traumatism—i.e., a blow on the kidney is an occasional cause. (4) The persistent use of certain drugs, such as cantharides and turpentine. (5) Inflammation secondary to disease of the urinary tract below the kidney (see Ascending Pyelo-nephritis). (6) Pregnancy is a marked predisposing, and sometimes exciting, cause. (7) "Trench nephritis" was a variety met with amongst the troops in Flanders. (8) Sudden chill when the skin is perspiring.

Prognosis.—Acute Nephritis may terminate in (1) complete recovery in a few weeks, when treatment and hygienic surroundings are good. This is usual with children; with adults complete recovery is not common except when the disease accompanies tonsillitis. (2) Partial recovery. If the disease lasts longer than three months, it usually develops into the condition known as large white kidney (Chronic Parenchymatous Nephritis, § 322). (3) Death may occur from uræmia, from dropsy into the serous cavities, or from other complications. The chief complications are: (a) Uræmia; (b) inflammations of the serous membranes, such as pleurisy, pericarditis, or peritonitis, which are usually latent—i.e., attended by little or no pain; and (c) inflammations of the mucous membranes, such as bronchitis, gastritis, enteritis (causing diarrhœa); (d) œdema of the lungs or of the glottis; (e) cardiac dilatation and failure; (f) erysipelas, cellulitis, and various other skin diseases are very prone to attack patients with acute nephritis.

The prognosis, therefore, of acute nephritis is grave in proportion to (i.) the diminution of urine; (ii.) the development of uræmic symptoms; (iii.) the amount of dropsy present; and (iv.) the nature and severity of the complications.

Treatment.—The indications are to relieve the kidney by giving it as little to do as possible; to increase the action of the skin and bowels; and to lessen local congestion. Only water or 5 per cent. glucose should be allowed for 24 to 36 hours. When dropsy is marked, little water should be taken. Give milk and whey freely at first; then sugar, cream, carbohydrates, and fruit. To obviate the danger from exposure to cold, the patient should be kept in bed till all red blood cells have disappeared

from the urine. Cases of scarlet fever should be kept in bed during convalescence, because they are so apt to develop this disease. Diaphoretics,
such as liquor ammoniæ acetatis, antimonium tartrate, warm baths, wet
packs, and hot-air baths. This treatment may be applied by means of
a cage placed upon the bed, and connected with a spirit-lamp through an
iron chimney at the foot. Purgatives, such as pulv. jalapæ co. (30 grs.
to ʒii (2–8)), are indicated. Saline purgatives are especially useful when
there is much dropsy. Diuretics are contraindicated in the early stage
only. Potassium bicarbonate, citrate, acetate, and bitartrate may be
given. In mild cases the Imperial drink may be taken freely—ʒiss.
cream of tartar dissolved in a pint of boiling water, flavoured with sugar
and lemon-peel. Scoparium and digitalis are given with caution if the
heart is feeble. Local depletion by wet or dry cupping is especially indicated when the urine contains much blood. Counter-irritation over the
kidneys, with poultices, antiphlogistine, or leeches, has a similar effect.
During *convalescence* tonics, especially iron, must be given. An admirable
prescription is liq. ferri perchloridi, ♏xv. (1) ; liq. ammoniæ acetatis,
ʒi. (4) ; acid. acetici, ♏v. (0·3) (to prevent decomposition). Milk diet
and a low diet must not be continued too long if the case becomes one
of chronic parenchymatous nephritis (§ 322). In the treatment of renal
disease three drugs are contra-indicated—opium, cantharides, and turpentine. Mercury is generally added to these, but I have never seen any
harm arise from its administration. For the treatment of Uræmia, see
§ 323.

Chronic Albuminuria.—There are three anatomical varieties of chronic
renal disease attended with more or less albuminuria, which, when occurring
in their typical forms, present well-marked clinical distinctions, as shown
in a tabular form below. In Chronic Tubal Nephritis (including large
white kidney), the renal *epithelium* is primarily and throughout the disease
chiefly involved. Chronic may follow acute nephritis, but in other cases
it is of insidious and gradual onset. In Chronic Interstitial Nephritis
the *interstitial tissue* shows evidence of increase, and throughout the disease
this is the most marked change ; the arteries also, however, show hyperplasia of their middle coat.[1] This arterial change also occurs throughout
the body, and is attended by a corresponding hypertrophy of the left
ventricle. In the Amyloid (or Waxy) Kidney the *vessels* are primarily
involved, the lardaceous degeneration beginning in the middle coat.
Pathologists make many subdivisions, but these represent the three
clinically recognisable groups of chronic renal changes attended by
albuminuria.

If the albumen is CONSIDERABLE, turn to Chronic Tubal Nephritis, Amyloid Kidney
(rare), or Chronic Renal Congestion. If there is only a TRACE of albumen, and the
urinary signs appear to be slight in proportion to the debility and other symptoms,
turn to Chronic Interstitial Nephritis, § 323.

The illness is **chronic,** *and the general symptoms of renal disease pro-*
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[1] *Cf.* Introduction to this chapter.

TABLE XX. DR. MURCHISON'S TABLE OF CHRONIC ALBUMINURIA.

	Quantity of Albumen.	Tendency to Uræmia.	Quantity of Urine.	Tendency to Dropsy.
Chronic Tubal Nephritis.	Large.	Moderate.	Diminished or normal.	Great.
Chronic Interstitial Nephritis.	Very small	Great.	Increased.	Very slight.
Waxy Kidney.	Very great.	Slight.	Greatly increased.	Slight.

nounced ; generalised DROPSY is marked ; the urine is scanty, and ALBUMEN and CASTS are abundant. The disease is CHRONIC PARENCHYMATOUS NEPHRITIS.

§ 322. Chronic Parenchymatous Nephritis (synonyms : Large White or Pale Kidney, Chronic Tubal Nephritis) may follow on acute nephritis, or may develop insidiously. In the latter stages the connective tissue is increased, and if the patient lives long enough the kidney becomes a *Contracted Fatty Kidney*, or *Small White Kidney*.

Symptoms.—(1) The albuminuria is considerable, $\frac{1}{4}$ to $\frac{1}{3}$ of the volume of the urine ; (2) the other characters of the urine are : (i.) the diurnal quantity is slightly diminished at first, but towards the end, when the kidney contracts, the quantity may be greater than normal; (ii.) the specific gravity is not much altered in the early stages, but the urea is deficient throughout ; (iii.) it is turbid, often with urates ; and recurrent hæmaturia may occur, especially if the condition has followed acute nephritis ; (iv.) all forms of casts are met with (§ 316). (3) There is generalised dropsy, but most marked in the face. It may disappear towards the end, when the diurnal quantity of urine increases. (4) There are pallor, emaciation, weakness, and digestive disorder ; and (5) cardio-vascular symptoms (§ 293) ensue.

Etiology.—Chronic parenchymatous nephritis follows (1) acute nephritis, or (2) prolonged mechanical congestion of the kidney (as in cardiac disease). (3) When it comes on insidiously, without apparent cause, seek some source of chronic infection of the blood, such as pyorrhœa, septic tonsils, ears, nasopharynx, or a diseased colon. (4) Alcohol in excess predisposes. (5) It is most often seen in males of middle age.

Diagnosis.—When the insidious form occurs in young women it is often mistaken for *chlorosis ;* in all cases of anæmia, examine the urine for albumen and the diastase and urea content. In the later stages it may be mistaken for *chronic interstitial nephritis ;* but in that disease the patient is usually older, and see Table XX. In certain cases which present *both* *renal and cardiac* symptoms, it may be very difficult to say *which condition is the primary* one. In such cases it is important to note the following points : (i.) If there is a *history* of rheumatic fever and previous attacks of dropsy, it is probable that the cardiac condition is primary. (ii.) If

other than mitral systolic murmurs are present it points to cardiac disease ; a mitral regurgitation murmur *alone* might be due to the cardiac failure following renal disease. (iii.) The *urine,* when there is any difficulty in diagnosis, is in both cases scanty and albuminous. Many tube-casts point to renal disease ; the rapid clearing up of the dropsy and improvement of the urine after a short period of rest in bed points to heart disease. (iv.) A *hard pulse* favours kidney disease, but an irregular soft pulse is found with cardiac failure secondary both to renal and to cardiac disease.

Prognosis.—The disease cannot be cured, and even with careful treatment the patient rarely lives many years. Death occurs with complications of uræmia, or as with acute nephritis. The prognosis is grave in proportion to (1) the amount of dropsy and albuminuria ; (2) diminution of urine and nitrogen excretion; (3) uræmic symptoms. Tests for renal efficiency should be made ; simple salt retention is not so grave as nitrogen retention. If a source of infection is found and removed, the outlook improves. When the stage of contraction sets in, life may be prolonged somewhat with care.

Treatment.—Chill must be avoided by warm clothing and life in equable climates. Moderate exercise is advisable. The bowels must act freely. For dropsy, Maclean finds a remarkable diuretic effect obtained by giving 15 gr. (1 gm.) urea dissolved in water twice a day. Till recently, such cases were restricted to a diet of milk and a minimum of protein, but it is now recognised that with this low diet œdema persists, and the patient has no energy. Many observers confirm Epstein's discovery that a proteid diet aids elimination of fluid from the tissues, and an improved sense of general well-being immediately follows the transition from a milk diet to one containing protein 120–240 grammes. Lean meat, ham, fish, white of egg, oysters, lentils, peas and beans, rice, bananas, oatmeal, skim milk, tea and coffee are allowed. Carbohydrates are limited to 150–300 grammes, and fats to 20–40 grammes, fluids to 1200–1500 c.c. per day.[1] Vegetables rich in oxalate, such as tomatoes, spinach, sorrel, rhubarb, beetroot and strawberries, should be avoided. It is best to avoid alcohol. Sufficient salt to render the food palatable is allowed ; a low salt intake aids the disappearance of œdema. When albuminuria and œdema persist for months in spite of careful treatment, and when cardio-vascular symptoms are absent, decapsulation of the kidneys may give good results.

The patient complains of lassitude, and other symptoms of INCIPENT URÆMIA *mentioned in* § 270. *There are only* TRACES OF ALBUMUMEN, *the diurnal quantity of urine is increased, dropsy is absent.* The disease is probably CHRONIC INTERSTITIAL NEPHRITIS.

§ 323. **Chronic Interstitial Nephritis** (synonyms : Contracted, Granular, or Gouty Kidney ; Cirrhotic Kidney ; Renal Fibrosis ; Small Red Kidney ; Chronic Non-Desquamative Nephritis).—It is accompanied by

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[1] Epstein, *Amer. Journ. Med. Sciences,* Nov. 1917 ; Maclean and de Wesselow, *Modern Methods in the Diagnosis and Treatment of Renal Disease,* 1921.

widespread cardio-vascular changes, as mentioned in the introduction to this chapter, consequent on recurrent high blood-pressure.

Symptoms.—(1) The albuminuria in this disease is small in amount, and many samples of the urine may be examined without finding any. In cold weather, however, when there is deficient skin action, there is generally a trace, especially after a chill or any cause which produces renal congestion. The other characters of the urine are : (ii.) The diurnal quantity is greatly increased (perhaps to 100 ounces). The patient often consults us because he has to get up at night several times to pass water. (iii.) The specific gravity is very low (1005 to 1012), owing partly to the deficiency in urea, but chiefly to the increased quantity of urine. The deficiency in total urea is not very great, and may not be sufficient to suggest the onset of uræmia ; the total amount of salts in the urine is diminished, and affords a more reliable indication (see § 311). (iv.) The urine is clear, pale, and contains but few casts, and these are chiefly hyaline or granular (Fig. 76). (2) Dropsy is usually absent. If dropsy occur it is due to (i.) secondary cardiac failure, or (ii.) the supervention of acute nephritis. (3) The patient may look robust, but sometimes he has a greyish pallor. (4) The pulse indicates persistent high blood-pressure, and is often associated with hypertrophy of the left ventricle, an accentuated aortic second sound, sometimes with a systolic apical murmur, and always sooner or later with a thickened condition of all the arteries. Later the heart may dilate, with consequent dropsy and albuminuria, and it may be hard to diagnose whether the kidney or the heart condition is primary or secondary (§ 322, *Diagnosis*). (5) There is throughout a condition of chronic or incipient uræmia (§ 296), due to the deficient nitrogenous metamorphosis in the body, and the retention in the blood and tissues of the antecedents of urea, owing to deficient renal function. These symptoms are indefinite, but in order of importance they are : (i.) Insomnia and *headache*, symptoms which, occurring in the aged, should always lead us to suspect granular kidney ; (ii.) gradual impairment of the mental and bodily vigour ; (iii.) tremors and twitching of the muscles ; (iv.) digestive disorders ; (v.) dyspnœa, often paroxysmal.

Course and Complications.—Apart from the existence of slight and intermittent albuminuria and persistent *high blood-pressure*, non-urinary symptoms are the earliest, and often for prolonged periods the only evidences of this disease. In many cases the symptoms of high pressure first reveal the disease to the physician ; in other cases it is the *ophthalmoscopic* changes (Renal Retinitis, § 293), changes which may or may not be attended by failure of vision. Apart from the progressive enfeeblement, the disease generally first manifests itself by the occurrence of one of its numerous complications. The most frequent *and most serious of these is cerebral hæmorrhage, resulting from the prolonged high blood-pressure and consequent arterial degeneration. Hæmorrhages of various kinds may occur in other directions, such as epistaxis, or melæna.* Epistaxis constitutes a kind of safety-valve, relieving the vascular system from

more serious internal hæmorrhages ; consequently it should not be checked. The *mucous membranes* are often affected, and intractable bronchitis or gastro-enteritis in an elderly person may be the condition which brings the patient under our notice ; the *serous membranes* less often, though a latent form of pleurisy or pericarditis is not uncommon. *Skin diseases* are often very troublesome. The earliest symptom noticed in many cases is the itching of the skin : Urticaria, eczema, erythematous, desquamative, and hæmorrhagic eruptions are apt to occur. The patient is liable, on exposure to cold, to attacks of *congestion of the kidney,* when the albuminuria and all the other symptoms are aggravated.

The *Diagnosis* from other forms of chronic renal disease is.given in Table XX, p. 439. However, the diagnosis of this form of chronic renal disease from the other conditions which give rise to lassitude and DEBILITY is often a question of much greater difficulty and is dealt with in § 401. In the diagnosis from cardiac failure due to cardiac valvular disease the history is important, and *cf.* p. 439, diagnosis.

Etiology.—(i.) I have assisted at an autopsy in a well-marked case of granular kidney in a child of nine ; but the disease almost invariably occurs in *persons of middle age or advanced life*. Out of 376 cases admitted into the Paddington Infirmary 317 were over forty years of age, 251 were over fifty, and 203 over sixty. (ii.) *Gout* and a gouty habit of body are the most important causal factors. In many cases there is a long history of persistent urates, and in a large proportion of cases of granular kidney the joints and ears show evidences of gouty deposit ; hence the name " gouty kidney." (iii.) An *indolent* life, and (iv.) *chronic lead poisoning* are undoubtedly causes, not only of gout, but of granular kidney. (v.) Various other forms of *toxæmia which produce recurrent or constant high blood-pressure* (*q.v.*) may also be followed by chronic interstitial nephritis. Many of the symptoms usually attributed to chronic interstitial nephritis (renal fibrosis) are really those of high blood-pressure, and in the author's opinion,[1] renal fibrosis is an accidental occurrence in certain cases of toxæmic high blood-pressure—not in all.

Prognosis.—The course of the disease, as already mentioned, is prolonged. With care and attention to diet the patient may live for five, ten, or more years, but the disease can never be cured. The amount of albumen is no criterion as regards prognosis in chronic interstitial, as it is in chronic parenchymatous, nephritis. The prognosis is grave in proportion (1) to the duration of the disease ; (2) to the evidences of uræmia present and their degree ; (3) the degree of cardiac failure ; and (4) the presence and severity of the complications (*vide supra*). Life is frequently terminated by cerebral hæmorrhage or some other complication ; a large number of these cases die of acute uræmia (§ 296), as the records of the Paddington Infirmary show. Older authors described this as death by " serous apoplexy," thinking that the serum which replaced the atrophy

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[1] " On Senile Epilepsy," the *Lancet*, July, 1909 ; and elsewhere. See also p. 899.

of the brain was the cause of pressure upon that organ.   Death may be
due to cardiac failure.

Treatment.—Chill must be avoided, and all undue exertion, mental
and physical.   Much that has been said regarding diet in § 322 is also
true of interstitial nephritis, but as the disease occurs in older persons it
is advisable to restrict the amount of food taken, solid and fluid.   The
blood urea content should be estimated from time to time, and when
this is increased the protein intake must be reduced.   Vegetable proteins
are preferable to flesh foods.   Fats and carbohydrates may be freely
taken provided they are readily digested.   Alcohol is prohibited, and
highly spiced articles of diet.   The cardio-vascular system requires careful
supervision.   High blood-pressure must be treated (§ 72) ; this relieves
headache and insomnia.   Purgatives such as Mist. Alba are good.   Iron
is of little use, and by leading to constipation may be injurious.   For
heart failure with•irregularity of the heart rest in bed with digitalis is
indicated ; when the blood-pressure remains high, give ammonium hip-
purate, gr. 5–10 (0·3–0·6), mannitol nitrate, gr. ½ (0·03), or sodium nitrite,
gr. 1 (0·65), and calomel in small doses daily or twice daily.   For renal
dyspnœa amyl or sodium nitrite and a mixture containing bromide, alkalies
and apt. ammon. aromat.   When with the cardiac failure there is salt reten-
tion and obstinate œdema, try rest in bed with the Karell diet : milk alone
for a week, 7 ounces given four-hourly for four feeds ; on the following
two days add egg and salt-free toast at two feeds, and on the eighth to
the twelfth day add chopped meat, rice, vegetables, and 1 egg.   The
treatment often resolves itself into the treatment of uræmia.   In *chronic*
uræmia keep the diet low, and endeavour to eliminate the poison by free
purgation, and encourage the action of the skin.   For *acute* uræmia—
muttering delirium, convulsions, coma (diagnosis of uræmic coma, § 564)—
a brisk hydragogue purgative must be given at once, such as pulv. elat.
co., pulv. jalapæ co., or a concentrated solution of magnesium sulphate.
The skin must be made to act by means of hot packs, hot air or vapour
baths, or pilocarpine, gr. ⅛ to ¼ (0·008–0·016) hypodermically.   Vene-
section (10 to 20 ounces) did a great deal of good in many of my infirmary
cases, and undoubtedly averted a fatal issue.   Transfusion of normal
saline solution compensates for the loss of fluid by bleeding or purgation,
and may be adopted after venesection.   Lumbar puncture with with-
drawal of 10 to 15 c.c. of fluid is beneficial and may arrest convulsions ;
chloroform relieves the convulsions.   For pulmonary œdema give atropin
in large doses, gr. $\frac{1}{50}$ (0·0012).   Acidosis is often present, and in such
cases give large doses of bicarbonate of soda (*cf.* § 393.)

There is abundant albumen *with the passage of* LARGE QUANTITIES *of urine, but
little tendency to dropsy and uræmia ; the patient has a history of prolonged* SUPPURATION,
or of SYPHILIS ; *and there may be evidences of lardaceous disease elsewhere.*   The disease
is LARDACEOUS KIDNEY.

§ 324. **Amyloid Kidney** (Waxy or Lardaceous Kidney) is generally part of a wide-
spread lardaceous disease involving the liver (enlargement), spleen (enlargement),

and intestines (diarrhœa). With more efficient modern surgical methods amyloid degeneration is becoming a very rare condition.

Symptoms.—(1) The albumen, though it may be small in quantity in the early stage, is very abundant, amounting to three-fourths or more when the condition is established. Apart from the albuminuria the urine is at first unaltered, but soon develops the characteristic changes : (i.) The diurnal quantity is greatly increased, even to 150 ounces ; (ii.) the specific gravity is very low, but the urea is not diminished till the later stages ; (iii.) the colour is pale and clear ; (iv.) all varieties of casts may be found, including amyloid and fatty casts. (2) There is great pallor of the surface and anæmia, but there may be no dropsy, till quite the end of the disease. In cases with great cachexia dropsy *may* occur early. (3) Evidence of lardaceous disease is present—enlargement of the liver and spleen ; consequently hæmorrhages may occur from different parts. Amyloid disease of the bowel gives rise to very intractable diarrhœa, a symptom which often accompanies amyloid kidney.

It is important for the *diagnosis* to ascertain the history of a *cause*—namely, (*a*) prolonged suppuration, either from a chronic abscess, chronic phthisis, or caries. Dr. Murchison used to be of the opinion that caries of the vertebræ, even without definite formation of an abscess, could give rise to lardaceous disease of the viscera, especially the kidney. (*b*) Syphilis is the second of the two great causes which bring about lardaceous disease.

Prognosis.—The course of the disease is protracted. The patient may live for several years, dying from exhaustion from diarrhœa, or other complications ; very-rarely from uræmia due to supervention of acute nephritis. With careful treatment patients may live for many years, or even recover if the disease is seen in a very early stage ; but the prognosis is bad in proportion to (1) the amount of albuminuria, and (2) the extent of the involvement of the other organs. The prognosis is very good if the septic focus is removed.

Treatment.—Alkalies have been reputed not only to prevent, but also to improve, the lardaceous process—*e.g.*, liquor potassæ, ℥ 5 (0·3) ; the tartrates and citrates of the alkalies are also administered. Iodine, especially in the form of iodide of potassium or iodide of iron, should be given, particularly in syphilitic cases. The most troublesome complication is diarrhœa. The only remedies which in my experience are of any use are liquor ferri pernitratis, ℥ 15 (1) ; or pil. plumbi cum opio, gr. 5 (0·3), continued every four hours until the diarrhœa ceases. Opium may be administered in this form of renal disease when there is no tendency to uræmia. The *preventive treatment* of lardaceous disease consists in the adequate treatment of syphilis in its early stages ; and in curing prolonged suppuration, especially when this occurs with chronic profusely discharging ulcers of the leg.

§ 325. In Renal Congestion (Secondary Albuminuria) there is sometimes a very considerable amount of albumen in the urine ; but the urinary and other symptoms do not conform to the foregoing types. Casts are generally absent, never abundant, and the constitutional disturbance, apart from the *primary* malady, is slight.

If the albuminuria is MARKED *and* CONSTANT, *and especially if the urinary symptoms are associated with symptoms referable to some other organ, it is probably* PASSIVE *renal congestion due to* I. CARDIAC DISEASE ; II. ASCITES or ABDOMINAL TUMOURS ; or III. PREGNANCY (?).

If the albumen is SLIGHT *in amount, and especially if it be* TRANSIENT, *it is probably* ACTIVE *renal congestion due to* IV. TOXIC BLOOD STATES, with or without PYREXIA ; V. DRUGS ; VI. DYSPEPSIA or HEPATIC DERANGEMENT ; VII. OBSCURE CAUSES ; VIII. FUNCTIONAL ALBUMINURIA or IX. CHILL TO THE SURFACE.

I. CARDIAC DISEASE (the Cardiac Kidney [1]) is the most frequent of the congestive causes of albuminuria. Albuminuria is a very common accompaniment of mitral valvular disease, and of the dilatation of the right heart which so frequently follows chronic bronchitis and emphysema. At first the kidney is only congested, but later the epithelium may become affected and the interstitial tissue increased. The diagnostic features of the albuminuria in such cases are : 1. The amount of the albumen is always considerable, and may be very great. 2. The urine is scanty, high-coloured, of high specific gravity, and there may be blood cells, renal cells, or even casts ; nevertheless these latter may disappear when the heart is relieved. 3. There are evidences of the cardiac condition which has produced the renal disease. In some cases it is difficult to decide which of these was primary (§ 322, *Diagnosis*).

Cardiac disease may give rise to renal disease in three ways : (i.) In the manner just stated. (ii.) *Embolism of the kidney* is one of the consequences of endocarditis (acute or chronic). In this condition the albuminuria appears suddenly with hæmaturia and constitutional symptoms, and disappears equally suddenly in a few days. (iii.) Some causes of *aortic valvular disease* (the mitral being healthy) have been attended by temporary albuminuria. The explanation is not obvious. In these cases the compensatory hypertrophy and dilatation were great, and the blood-pressure high ; and it seems probable therefore that the albuminuria may have been due to *active* renal congestion.

II. ASCITES AND ABDOMINAL TUMOURS.—Here the albuminuria is due to pressure on the renal veins. This condition is recognised by : (1) The amount of albumen is generally moderate ; (2) there is abdominal enlargement with the signs of fluid or tumour ; (3) the albuminuria will disappear on removing the cause. There are two fallacies to be remembered before diagnosing albuminuria as due to ascites : (i.) Both albuminuria and ascites may be the product of some common cause—*e.g.*, heart disease ; and (ii.) the ascites may be the result of a general dropsy due to renal disease.

III. PREGNANCY is an undoubted cause of albuminuria, and according to Playfair it occurs in 20 per cent. of parturient women after the third month. It also seems certain that permanent and ineradicable renal disease may, in some cases, date from pregnancy. According to some, the albuminuria of pregnancy is due to pressure on the renal veins—a view that is supported by its more frequent occurrence in primiparæ, in whom the abdominal walls are more rigid. But, on the other hand, the albuminuria may occur before the uterus is large enough to cause pressure on the renal veins. These and other considerations point to the conclusion that it is probably due to some blood change associated with the parturient state. The clinical features are : (1) The amount of albumen

[1] It is well to bear in mind that when both cardiac and renal disease are present, they may be associated in three ways : (*a*) Cardiac disease may produce renal disease in one of the above-mentioned ways. (*b*) Renal disease may produce cardiac disease, as when acute nephritis or granular kidney lead to cardiac hypertrophy and failure. (*c*) They may both be the result of a common cause—*e.g.*, gout.

is not usually great, and the urine is otherwise normal or very much as in cardiac cases. (2) Ophthalmoscopic changes (§ 293) may be present; but (3) these and the urinary symptoms disappear within two or three weeks of labour unless permanent renal disease has been induced. The treatment is discussed below.

The remaining causes of albuminuria are probably due to ACTIVE congestion of the kidney.

IV. TOXIC BLOOD STATES with or without PYREXIA.—This cause of albuminuria is characterised by : (1) Tube-casts are absent unless there be active renal disease. (2) An elevated temperature; in hyperpyrexia albuminuria is invariably present. (3) Other evidences of the toxic blood state, namely : (i.) Various acute specific fevers—*e.g.*, diphtheria, where albuminuria may be present without high temperature. In scarlet fever albuminuria frequently comes on between the sixteenth and twenty-sixth day, at which time also acute nephritis may supervene, and, to avoid this risk, scarlet fever patients should be kept in bed three or four weeks. Transient albuminuria may occur in secondary syphilis, between the sixth and eighth weeks of the disease. If albuminuria occurs in the later stages of the disease, it may be due to lardaceous disease or gumma of the kidney. (ii.) Acute pneumonia is sometimes, and (iii.) acute gout is very frequently, accompanied by albuminuria. (iv.) Albuminuria may also occur in diabetes (in which it is a grave sign), in any severe anæmia, and the reaction stage of cholera.

V. Various DRUGS, such as morphia, quinine, phosphorus, arsenic, cantharides, cubebs, copaiba, turpentine, salicylic acid, mercury, and carbolic acid, may give rise to albuminuria. This cause is recognised by (i.) the presence of the drug in the urine ; (ii.) there may be a history of the administration of the drug ; and (iii.) the albuminuria usually disappears when the drug is stopped.

VI. DYSPEPSIA and LIVER DERANGEMENT are sometimes accompanied by albuminuria. The symptoms of hepatic congestion may be present, showing the intimate connection between the hepatic and renal functions (§ 263). Albuminuria is sometimes present with that form of dyspepsia which is accompanied by oxaluria. Certain articles of diet are known to have been attended by albuminuria. Thus, cases have been recorded in which albuminuria followed the ingestion of shell-fish, eggs in excess, cheese, and large quantities of alcohol. In many of these instances the condition is probably albumosuria (§ 312).

VII. OBSCURE CAUSES, *e.g.*, when albumen appears for unknown reasons, as (1) after burns and other causes of severe shock. (2) In exophthalmic goitre the albuminuria is usually temporary, though it may last for months. It may vary in amount at different times on the same day, which tends to show that it is of vaso-motor origin. The urine in other respects is healthy. (3) Excessive study or other cause of nerve strain has been reported to have occasioned albuminuria. (4) Certain cases of cerebral tumour, and other conditions in which there is increased intracranial pressure, have been attended by albuminuria. (5) Albumen is found in the urine after epileptic fits.

VIII. PHYSIOLOGICAL OR FUNCTIONAL ALBUMINURIA.—A cyclic, postural, ortho-static or adolescent form of albuminuria, or "leaky kidney," has been described. It appears regularly at some time each day, usually in the morning or after a cold bath. It is usually absent at night, or when the patient retains the horizontal position. It occurs in subjects with vasomotor instability. Albuminuria has been found in schoolboys and athletes after violent exercise ; it may last for several hours, but disappears after the night's rest. *Paroxysmal* albuminuria is probably closely related to paroxysmal hæmoglobinuria (below) ; it appears at intervals, without any apparent cause, and lasts for a few days or weeks at a time. Some of the reported cases were probably early stages of Raynaud's disease, others were perhaps associated with oxaluria. In functional albuminuria the diastase output is normal, whereas it is

diminished in chronic nephritis and increased in toxic nephritis ; the globulin ratio in increased.

IX. CHILL TO THE SURFACE.—Chill to the surface may result in albuminuria, but in such cases the kidney is rarely quite healthy. This condition is recognised by : (1) The amount of albuminuria is never very great, and it does not last for more than a few days ; (2) the urine is otherwise normal, or may deposit urates ; (3) the patient, in other respects, is healthy, or complains only of slight bronchial catarrh or coryza.

The *Prognosis* of albuminuria due to congestion is very much that of its cause. Before giving a prognosis it is important to thoroughly and repeatedly examine the urine, for casts in particular, so as to be satisfied that the kidneys are structurally healthy. Young subjects of functional albuminuria, excluding that form which follows athletic exercise, are not necessarily predisposed to kidney troubles, but they are often under par ; the albuminuria may disappear in three to seven years. The prognosis as to life is excellent.

Treatment.—The treatment must be directed to the cause. Rest in bed will do a good deal for the renal complication of cardiac disease. In the *albuminuria of pregnancy* careful investigations should be made, and the amount of urea watched. If (1) there is a clear history of renal disease prior to pregnancy, or (2) puerperal eclampsia has occurred in previous pregnancies, or (3) the renal disease, no matter of what kind it may be, is distinctly *progressive in its nature*, then premature labour should be induced. For the treatment of functional albuminuria general hygienic and dietetic rules must be followed. The administration of calcium lactate, by increasing the coagulability of the blood, temporarily stops the albuminuria.

§ 326. Hæmaturia.—When the patient is " passing blood " in the urine, an endeavour should be made to ascertain if the blood comes chiefly at the beginning of micturition, chiefly at the end, or whether it is intimately mixed with the urine and gives to it a " smoky " tint. For the test for blood in the urine and the methods of distinguishing it from hæmoglobinuria, see § 309. The fallacy of menstrual blood must be avoided by using a catheter.

A. *If the blood is bright crimson and comes chiefly* AT THE COMMENCE-MENT *of micturition, it is probably of* URETHRAL *or* PROSTATIC *origin.*

In these circumstances, which are mainly of surgical interest, there will probably be a history of injury or gonorrhœa. In congestion or abscess of the prostate there are local pains or tenderness and rectal irritation. Urethral angioma and excessive sexual indulgence may lead to hæmaturia in the male.

B. *If the blood comes most freely* AT THE END *of micturition, and especially if in clots, it is probably of* VESICAL *origin.*

The COMMONEST CAUSES of vesical hæmorrhage are :

I. ACUTE CYSTITIS, chiefly at its onset (see § 330). The hæmorrhage is usually slight.

II. CALCULUS, or stone, in the bladder. Here the hæmorrhage is worse after exercise, moderate in amount, and there is pain, which, like the bleeding, is worse at the end of micturition and after exercise, and is frequently referred to the point of the penis. The ensuing cystitis may complicate the symptoms and render the diagnosis of stone difficult, but its detection by the sound or cystoscope is conclusive.

III. TUMOURS of the bladder.—The hæmorrhage here, especially in *villous* tumours, is usually great in amount. Shreds of the growth may be passed, and cystitis may develop. In *cancerous* tumours the hæmorrhage is more or less intermittent and

resists treatment; there are pain and cachexia, and sometimes the growth may be palpable above the pubes or per rectum. Extension of tumours of neighbouring organs, or even spread of inflammation or congestion, as in appendicitis or dysenteric ulcers, may cause hæmaturia. The cystoscope is the best means we have of recognising the condition of the bladder.

Some of the LESS COMMON CAUSES of vesical hæmaturia are TUBERCULOUS DISEASE of the bladder (when the bacillus may usually be found), VESICAL VARIX, certain constitutional diseases such as SCURVY and PURPURA, and SCHISTOSOMA HÆMATOBIUM.

SCHISTOSOMIASIS, "Endemic Hæmaturia," occurs chiefly in Egypt and South Africa. Schistosoma hæmatobium (Bilharzia hæmatobia) is a trematode found in the portal system of man; the ova are set free in the venous plexuses about the bladder and are passed in large numbers in the urine. The ova are also carried backwards in the blood-stream and have been found in almost every organ of the body; the irritation they cause gives rise to serious fibrotic changes. The ovum (Fig. 87) is recognised by its characteristic terminal spine. Damage by this spine causes hæmaturia. Schistosoma mansoni and japonicum are mentioned in § 249. The ova of all three species of trematode continue their development outside the human body if they reach water. An actively motile miracidium is set free and penetrates certain fresh water snails in which multiplication takes place. The parasites are once again set free in the water and re-enter the human body through the skin or mucous membranes during the act of bathing or when imbibed. The hæmorrhage may be very great, and severe anæmia result; or the disease may persist with only slight signs for years. The presence of eosinophilia may suggest the nature of the disease when occurring in a country where it is rare.

The *Diagnosis* and *Treatment* until recently lay almost entirely in the hands of the surgeon. With the introduction of the method of treatment by intravenous injections of tartrate of antimony there is hope of ridding Egypt of this dangerous scourge. A dose of 2½ grains is given every second day. Usually 25 grains is sufficient to destroy the parasite and the ova; as many as 60 have been given. The dead ova show as dark shrivelled bodies with granular contents; they may continue to be eliminated for a year or more after the injections. As the antimony kills the ova as well as the worm the patient ceases to be a carrier—a fact of great prophylactic importance.

FIG. 87. — EGG of Schistosoma hæmatobium, magnified about 100.

C. If the blood is INTIMATELY MIXED with the urine, causing it to assume a "smoky" tint, it is probably of RENAL origin. In these cases also the tests for blood should be carefully applied, and fallacies avoided (§ 309).

Symptoms and signs pointing to the kidney will usually be detected on examination.

The CAUSES of RENAL HÆMORRHAGE may for convenience be grouped into : Inflammation ; calculus and other causes of pyelitis ; local conditions ; causes from distant parts ; paroxysmal hæmorrhage ; and parasites.

I. In acute nephritis the blood usually gives rise to the characteristic "smoky" urine, and the deposit contains casts (§ 321). II. Renal calculus (see below). III. Tuberculous disease of the kidney (§ 331). IV. Any of the other causes of pyelitis (§ 331) may give rise to red corpuscles in the urine in larger or smaller amounts. V. Malignant and other tumours of the kidney, especially cystic kidney, cause profuse and sometimes intermittent hæmorrhage (§ 341). VI. Villous disease of the pelvis of the kidney. VII. Injury to the kidney (below). VIII. Passive congestion of the kidney—for example, in heart disease or chill. IX. Embolism of the kidney (see Endocarditis, § 45). X. Blood poisons and blood diseases—acute fevers, scurvy, purpura, malaria, leukæmia, etc. XI. Coli infection and oxaluria. XII. Drugs,

such as cantharides, turpentine, and phosphorus. XIII. Paroxysmal hæmoglobinuria (§ 328) differs from all the foregoing in the absence of blood discs, though blood colouring matter is plentiful in the urine. XIV. Parasites—*e.g.*, Schistozoma hæmatobium (see above). The micro-filaria sanguinis hominis usually causes chyluria, but hæmaturia also may occur.

§ **327. Renal Calculus and Renal Colic.**—Calculi may form either in the pelvis of the kidney or, more rarely, in its substance. Perhaps the commonest form consists of *uric acid* and urates mixed in varying proportions (§ 307). These form stones of light brown colour, either round or branching, and are the commonest stones in subjects of the gouty diathesis, and those whose highly acid urine habitually deposits urates. Another variety, dark brown in colour, consists of *oxalate of calcium*, and gives rise to acuter symptoms, for each bristles with sharp-pointed crystals. Calculi are often multiple. Compound stones occur, consisting of an oxalate or organic nucleus, or alternate layers. Phosphates are uncommon; cystine and xanthin are only rarely met with in renal calculi. Various *events* may happen. (1) A calculus may remain in the renal pelvis, giving rise to chronic pyelitis (§ 331) for years; or (2) by its movement produce acute symptoms, RENAL COLIC. (3) It may obstruct the ureter and lead to hydro- or pyo-nephrosis (§ 341). (4) If the other kidney is not healthy sudden blocking may lead to obstructive suppression (§ 338). (5) It may pass into the bladder and result in cystitis. (6) Small stones may be voided through the urethra as " gravel." (7) In rare cases small calculi become encysted and quiescent. The typical clinical history of renal calculus consists of (*a*) *attacks of renal colic*, separated by (*b*) *intervals* in which the symptoms are those of calculous pyelitis (§ 331).

The *Symptoms of Renal Colic* consist of severe paroxysms of lancinating pain, starting in one loin, shooting down to the testicle or vulva on that side; attended by vomiting, shivering, sweating, pallor, and a certain amount of collapse. These symptoms are in most cases followed by hæmaturia, the urine containing blood discs and pus cells, but usually no casts. Crystals are also present, and guide us as to the nature of the stone. It is with the oxalate calculus that most blood and pain occur. The *diagnosis* of renal from other forms of colic is given in table XIII, § 196. Cystoscopic examination may reveal irritation, or blood issuing from one ureteral orifice. All the symptoms of renal colic may arise simply from the irritation of *fine crystals*. They may also be produced without alteration in the urine by *movable kidney ;* or by the passage of *clots* of blood or caseous material down the ureter. *Malignant* disease of the kidney may be mistaken for calculus, but in that case the blood is more copious and more constant, and the pain is less severe, but more continuous. X-ray examination is of assistance except in the case of uric acid and cystine stones.

Treatment.—(1) Of the colic and (2) during the intervals. 1. The treatment of an attack of *renal colic* consists mainly in the relief of the symptoms—pain, vomiting, and collapse. Usually nothing avails except

inhalations of chloroform, and injections of morphia or papaverine may safely be given unless there is reason to fear the renal parenchyma is also diseased. Locally hot applications relieve. Effervescing citrate of potassium with spiritus ammoniæ aromatici may be administered with advantage. Between the painful attacks the patient must rest to allow the subsidence of inflammation. 2. The treatment in the intervals resolves itself into the solution or removal of the stone, and treatment directed to the pyelitis. Dietetic treatment is of great use in some cases. In uric acid cases a purin free diet is given (§ 274). If oxalates are being passed, any dyspepsia should be carefully treated ; such articles of diet as rhubarb, tomatoes, cabbages and onions, sweets and alcohol, should be avoided. The urine in all cases should be kept diluted by drinking plenty of fluid. The alkaline waters are very useful here, such as those of Vichy, Ems, and Contrexéville. In uric acid ˉcalculus, large doses of alkaline salts are certainly useful, especially the citrate and the acid tartrate of potassium. Begin with gr. 50 (3·3) of potassium citrate in ℥ iv. (128) of water every four hours until the urine is alkaline, and then give an effervescing drink, consisting of ℨi. (4) of sodium bicarbonate, and grs. 40 (2·6) of citric acid in 4 ounces (128) of water, three times a day. This treatment should not be continued if the urine is or has become ammoniacal. For pyelitis see § 331. Operative treatment is called for if repeated attacks of colic recur, or if the stone can be detected by radiographic examination.

Injury of the Kidney, laceration or rupture, is usually caused by a fall on the back of loin, or in " buffer accidents " on the railway during shunting operations. There may be no bruising or external signs, but a laceration of the kidney may be inferred from (1) the history of such an accident ; (2) a tense swelling (due to extravasated blood) with increased area of dulness in the region of the kidney ; and (3) copious hæmaturia. In a few cases there is no hæmaturia, and the other two evidences have to be relied on. Immediate operation is advisable, the collapse being treated by saline injections.

§ 328. In Paroxysmal Hæmoglobinuria porter-coloured urine is passed at intervals. An attack commences abruptly with (1) *a rigor* or " chilliness," nausea, and malaise ; and (2) lumbar pain. (3) An hour or so later the patient passes dark, highly albuminous urine, showing the spectroscopic band of methæmoglobin or of hæmoglobin, containing no red discs, but a quantity of amorphous granular matter. It has a specific gravity of 1020 to 1022, a slight excess of urea, and deposits crystals of oxalate of calcium. Each attack lasts a few hours, and passes off as suddenly as it came, but only to recur in a few hours' or days' time. In the intervals the general health is fair, but later the patient becomes anæmic and languid. Relapses recur for months or years without fresh exposure to " chill."

The *Causes* are obscure. The symptoms indicate destruction of blood in the vessels, with a setting free of hæmoglobin which is eliminated by the kidneys. In 90 per cent. of the cases (Roberts) the attacks are connected with chill to the surface. The disease is sometimes associated with Raynaud's disease, rheumatism, syphilis, malaria, mental or physical over-exertion, and dyspepsia. In most cases there is a positive Wassermann. It may occur in families.

The *diagnosis* of this tendency can be made by lightly ligaturing a finger and immersing it in cold water ; in these subjects such a degree of exposure to cold is enough to cause hæmolysis of the blood in the finger, with subsequent slight hæmoglobinuria.

The *Treatment* consists of rest in bed during the attacks, with warmth, and hyoscyamus internally. Persons predisposed to such attacks should avoid exposure to cold.

HÆMOGLOBINURIA may occasionally accompany Raynaud's disease, severe burns, and acute infective diseases, especially malaria. For Blackwater fever, see § 409.

HÆMOGLOBINURIA may be produced by toxic doses of chlorate of potassium, naphthol, pyrogallic acid, carbolic acid, arseniuretted hydrogen, carbon monoxide, and quinine in those who have had malaria.

EPIDEMIC HÆMOGLOBINURIA is seen in the new-born, with jaundice and nervous symptoms.

The patient complains of LASSITUDE *and ill-health, which have come on gradually ; the urine is found to contain* PUS *(§ 414)—i.e., there is* PYURIA. *With few exceptions (see footnote p.* 452), *when the pus comes from the* BLADDER *the urine is* ALKALINE, *and the pus remains diffused through the urine ; but when it comes from the* KIDNEYS *or any other part of the urinary passages the urine is* ACID, *and the pus settles at the bottom. Pus cells produce a trace of albumen in the urine.*

§ 329. **Pyuria.**—If we except the rupture of an abscess into the urinary passages, there are three sources of pus in the urine :

A. From the **Urethra** (*e.g.*, gonorrhœal or B. coli infection).

B. From the **Bladder** (cystitis).

C. From the **Kidney** (pyelitis).—The chief forms of pyelitis are CALCULOUS, TUBERCULOUS, ASCENDING and that due to B. COLI INFECTION.

Abscesses bursting into the Urinary Tract.—The abscesses most liable to burst into the urinary tract are : (*a*) prostatic abscess (below); (*b*) perineal abscess ; (*c*) pelvic cellulitis ; (*d*) psoas abscess ; (*e*) perinephric abscess ; and (*f*) abscess of the liver ; and there are also many other sources. (i.) The urine is usually acid ; (ii.) the pus is in large quantity and settles at the bottom ; (iii.) there is a clinical history of abscess prior to the appearance of pus in the urine ; and (iv.) localising signs of the abscess may be present.

It is believed by some observers that persons in health may pass a few leucocytes, but it is extremely probable that these are always derived from the generative organs (male or female), and that the occurrence of *any* pus cells in a properly collected catheter specimen is always pathological.[1]

When the presence of pus is suspected, the reaction should be tested immediately after it is passed, before decomposition can set in. Decomposition makes the urine ammoniacal, and therefore alkaline.

A. *The* **pus** *comes chiefly at the* BEGINNING OF MICTURITION, *and the urine is* ACID ; it comes from the URETHRA, and is usually caused by one of three conditions :

I. URETHRITIS.—There is pain, swelling, and redness of the meatus, scalding during micturition, and discharge of pus apart from micturition.

II. PROSTATIC ABSCESS is known by : (1) pain at the end of micturition ; (2) the finger in the rectum detects a tender, fluctuating swelling ; (3) the symptoms closely resemble those of vesical calculus with concurrent cystitis. It may be distinguished

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[1] In some cases there is a history pointing to leucorrhœa or gleet, but the quickest way of settling this point is to draw off the urine by catheter.

from this, however, by : (i.) a history of gonorrhœa, which is the chief cause of prostatic abscess ; (ii.) the signs on examination per rectum ; and (iii.) a discharge occurring in the intervals between micturition.

III. PERINEAL ABSCESS is detected by the local signs.

B. *The pus comes chiefly at the* END OF MICTURITION, *or is intimately mixed with the urine, which is* ALKALINE *when tested ˙immediately after it is passed.*[1] The pus comes from the BLADDER, and is indicative of CYSTITIS.

§ 330. **Cystitis,** or inflammation of the bladder, occurs in two well-, recognised forms—acute and chronic.

(*a*) In ACUTE CYSTITIS.—(1) In this condition the pus is in small amount, and in severe cases there may be considerable hæmaturia at the outset. At first the urine is acid, but it soon becomes alkaline, and ropy with pus and mucus. (2) There are pain and tenderness in the hypogastrium. (3) Micturition is frequent and painful ("˙ scalding "). After micturition the pain is relieved for a short time, unless the cystitis is due to stone in the bladder, when the pain is severe *after* micturition, because the inflamed walls of the emptied bladder then come into contact with the stone. (4) There is generally marked constitutional disturbance, with pyrexia.

(*b*) In CHRONIC CYSTITIS (which may supervene upon the acute form, or may be chronic from the outset), there is (1) a larger amount of pus. (2) The urine is alkaline directly it is passed and contains a large amount of ropy mucus (*cf.* footnote below). (3) The pain and other symptoms are less severe than in acute cystitis.

Etiology.—(i.) Gonorrhœa causes the most severe and often fatal form of acute cystitis and pyelo-nephritis. Other causes are (ii.) stone or foreign bodies setting up irritation ; (iii.) injury by instruments or foreign bodies introduced by the patient ; (iv.) the use of catheters which have not been rendered thoroughly aseptic ; (v.) cancer, villous disease, and other tumours of the bladder ; (vi.) urine decomposing in the bladder, as in stricture urethræ, prostatic enlargement, and other causes of retention of the urine (§ 337) ; (vii.) various nerve complaints producing paralysis and retention ; (viii.) extension from a urethritis or inflammation from adjacent organs, as in pelvic cellulitis ; (ix.) tuberculous disease of the bladder ; (x.) other microbes, notably the bacillus coli communis, produce cystitis which may be preceded by pyelitis (§ 331) ; (xi.) drugs—*e.g.,* cantharides or turpentine.

Differentiations.—(1) Cystitis due to VESICAL CALCULUS.—¹In addition to the symptoms of simple cystitis, there are (i.) pain at the end of micturition, lasting for some time after, very severe, shooting down the urethra ; (ii.) hæmaturia is common, though in some cases it may be so slight that it is detected only by the microscope ;

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[1] At the outset of acute cystitis the urine may be acid, and it may become acid again in the stage of recovery from chronic cystitis. It may also be acid in the early stage of tubercle and new growths of the bladder, and in cases of cystitis due to bacillus coli communis. In all other conditions in which the urine contains pus derived from the bladder the reaction is alkaline.

(iii.) a history of renal colic (§ 327) ; (iv.) the stone may be detected by the sound or the cystoscope.

(2) Cystitis due to NEW GROWTH IN THE BLADDER, or ULCERATION, is characterised by (i.) paroxysms of lancinating pain, quite independent of micturition and movement ; (ii.) copious hæmorrhage at intervals, occurring without apparent cause ; (iii.) the urine may contain cancer cells or tubercle bacilli ; a tumour may be felt per rectum or through the abdominal wall. (iv.) Cystoscopic examination may settle the diagnosis.

Prognosis.—Cystitis is not dangerous to life unless the inflammation spreads upwards from the bladder to the kidneys and produces pyelonephritis ; but, on the other hand, it is a very troublesome, painful complaint, and has a special liability to recur. When the cause is not removable--*e.g.*, in cystitis due to tumours of the bladder—the prognosis is very grave. When it is due to retention of urine (such as that caused by the atony of the bladder in old age), and when it is due to gonorrhœa, it tends to cause ascending pyelitis and pyelo-nephritis. When there is pre-existing hydronephrosis (§ 341), and acute cystitis develops, the inflammation is almost certain to extend upwards to the kidney, and so lead to pyonephrosis.

Treatment.—The cause must be sought for, and, if possible, removed. (*a*) In the *acute* form absolute rest in bed with milk diet is necessary. Copious libations of water, barley-water, and other bland fluids are called for. Alkalies, especially potassium citrate, and buchu and uva ursi are useful. Mild laxatives should be given, combined with hyoscyamus. Hot sitz-baths and morphia suppositories relieve the pain. (*b*) For the *chronic* and *subacute* (non-tuberculous) forms it may be necessary to wash out the bladder with hot water and boric acid. It is better to use a strong solution of boric acid, not exceeding 2 ounces at each sitting, than to wash out with large quantities. When the bacillus coli is present, give acid phosphate of soda, gr. xx. (1·3), followed by hexamin, gr. xx. (1·3), till the urine is scalding from the formation of formalin in the bladder. Hexamin is not a really efficacious urinary germicide unless given in this way. When streptococci and staphylococci are present, boric acid and benzoate of soda are the best drugs. (*c*) Autogenous vaccines are often of great service in uncomplicated bacterial infection of the urinary tract, especially tuberculous and coli infections.

C. The **pus** *is associated with a urine which is* ACID *when freshly passed* (*acid* **pyuria**), *the pus cells are at first disseminated through the urine, but in a short time they settle down as a* SEDIMENT, *and there is* PAIN, *and perhaps,* SWELLING *of the kidney* ; the pus comes from the kidney—the disease is PYELITIS.

§ 331. **Pyelitis,** or inflammation of the pelvis of the kidney, is indicated by the symptoms just mentioned. The urine, which is acid unless there be concurrent cystitis, contains, in addition to pus cells (Fig. 79), epithelial cells from the renal mucosa ; but, unless the renal parenchyma is involved, no casts and no albumen in excess of the quantity which would be accounted for by the pus are found, nor is there any dropsy. There is increased

frequency of micturition. Renal pain (nephralgia) and tenderness are nearly always present, but they vary widely in degree and character in the three varieties about to be mentioned. *The kidney should always be carefully examined* (§ 318), because, in addition to the renal congestion, all forms of pyelitis are liable to result in partial or complete obstruction of the infundibula, and the gradual supervention of hydro- or pyo-nephrosis. A few pus cells in the urine may be found in acute nephritis, after enteric and other fevers, and toxic doses of cantharides or turpentine. Apart from these there are three well-marked varieties or causes of acid pyuria.

I. CALCULOUS PYELITIS is due to the irritation set up by the presence of a stone. The *Differential Symptoms* are : (i.) A history of renal colic (§ 327) is often obtainable. (ii.) *Pain on one, the diseased, side*, which varies with exercise, and (iii.) hæmaturia, also varying with exercise. (iv.) The quantity of pus often varies from day to day, and the patient may feel easier after a discharge of pus, as the retained pus causes pain, and sometimes swelling. (v.) Attacks of intermittent pyrexia and sometimes rigors, from time to time. (vi.) *Crystals in the urine* aid the diagnosis considerably.

II. TUBERCULOUS PYELITIS.—Tuberculous disease of the kidney may be primary or secondary to tubercle elsewhere. Very often both kidneys are diseased. This condition may be very difficult to diagnose from Calculous Pyelitis, but the *Differential Symptoms* are : (i.) No previous history of colic, but dull pain in the loins, liable to exacerbations from the passage of caseous masses ; (ii.) hæmaturia is not usually present ; [1] (iii.) the amount of pus in the urine does not vary but steadily increases ; (iv.) the urine contains amorphous granular matter and tubercle bacilli, but usually no crystals or tube-casts ; (v.) *pyrexia of a regularly intermitting type*, with increasing emaciation ; and (vi.) there are often evidences of tubercle in other parts of the body, as in the testes or lungs ; (vii.) the cystoscope may show the presence of swelling or ulceration at the mouth of one ureter ; (vii.) Calmette's and Von Pirquet's reactions are present, and the opsonic index indicates tubercle (*vide* § 417).

III. ASCENDING PYELITIS or PYELO-NEPHRITIS arises from three groups of causes, which may conveniently be termed Obstruction, Extension, and Infection Pyelitis. (*a*) Some *obstruction in the urinary passages* below the kidney not infrequently causes retention and decomposition of the urine, and septic infection of the pelvis of one or both kidneys, which may go on to pyo-nephrosis. The diagnosis of this form of pyelitis, which used to be known as " Surgical Kidney," rests mainly on the history of the cause of retention—enlarged prostate, urethral stricture, uterine and other tumours pressing upon, or calculus impacted within, the ureter (see also Retention, § 337). Here, as in the next group, the urine may be alkaline from concurrent cystitis. (*b*) Ascending pyelitis may also result from the *extension of cystitis* without obstruction, and thus the numerous causes of the latter disease (§ 330) are brought into operation—*e.g.*, gonorrhœa, septic catheterisation, etc. (*c*) INFECTIVE PYELO-NEPHRITIS.—Previous bladder symptoms may be slight, transient, or altogether absent ; the bacillus coli communis is the most frequent cause

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[1] Occasionally hæmaturia is an early symptom of renal tuberculosis.

of the condition. On the other hand there may be frequent painful micturition, leading to a mistaken diagnosis of cystitis, especially in women. Mr. Frank Kidd considers that in the majority of such cases the kidney is primarily infected from the blood stream by the bacillus coli. He finds that frequent and painful micturition, with fever, is more often due to coli pyelitis than to cystitis. The disease occurs chiefly in females, as children or during pregnancy, but may occur at any time. The right kidney is more often affected than the left. The *Symptoms* of coli infection may be wholly indistinguishable from calculous or tuberculous pyelitis, unless one is aided by the detection of the respective microbes in the urine. In some cases there is a communication between the urinary tract and a septic focus, such as a pelvic abscess. There are three features which in the author's experience are characteristic of the coli infection : (i.) the occurrence of attacks of pyrexia at irregular intervals of a distinctly pyæmic type, attended by shivering, sweating, vomiting, and pain in the kidney ; (ii.) a distinctive smell of volatile sulphides in the urine ; and (iii.) the fact that pure cultures of b. coli can be readily obtained from a specimen of the urine collected through a sterilised catheter.

Bacilluria is the term employed to indicate the condition when the symptoms are more indefinite, less indicative of involvement of the kidney. It is one of the causes of hectic fever and debility in children, and is only diagnosed on the detection of the bacilli.

Prognosis.—(i.) The most serious form of pyelitis is that due to extension of inflammation upwards from the bladder. When originating in gonorrhœal cystitis, death usually occurs in seven to fourteen days. (ii.) In the tuberculous form there may be no general symptoms until the disease extends beyond the one kidney ; in other cases it may be fatal in twelve to eighteen months. (iii.) Calculous pyelitis may last indefinitely for years, though not without danger of uræmia and abscess of the kidney. (iv.) The course of ascending pyelitis depends very much upon the cause, the possibility of its removal, the age of the patient, and his general condition. Coli pyelitis runs an indefinite course. There have been deaths ; the majority recover, but the condition tends to recur. *Pyonephrosis* (§ 341) may ensue in all the chronic forms of pyelitis.

Treatment.—In all forms of pyelitis fluid diet and warm drinks, absolute rest in bed and warmth are essential ; cupping of the loins is sometimes useful. 1. The most common form, frequently mistaken for cystitis, is that occasioned by bacillus coli infection. When there is fever, and bacillus coli is found in the urine, the best drug is citrate of potassium gr. 40 to 60 (2·6–4). This large dose can be given every three or four hours, either alone or combined with other alkalies (potassium, sodium and magnesium carbonate) until the urine is alkaline to litmus paper. The alkaline treatment is then continued, in smaller doses, until the temperature has been normal for several days. Then hexamine, gr. 10 to 20 (0·65–1·3), is given after rendering the urine acid with large doses of acid sodium phosphate, and continued for a month. In chronic cases small doses of autogenous vaccine are very successful. During the acute stage, water and barley water must be drunk freely ; four to six pints daily may be taken. Certain chronic cases respond best to kidney lavage with 20 per cent. colloidal silver (Frank Kidd). 2. Many cases of pyelitis call for nephrectomy or for surgical measures. Before operation it is

necessary to determine which kidney is diseased; this is done by the
cystoscope, the ureteral catheter and sometimes by X-ray examination.
In cases of *calculous* pyelitis, large doses of potassium citrate and bicar-
bonate may be employed for uric acid calculi; for oxalates, nux vomica,
and nitro-hydrochloric acid; and nephrolithotomy in nearly all cases.
3. In cases of *tuberculous* pyelitis. Tonics such as iron, quinine, and cod-
liver oil must be given. Excision of the kidney is to be advised if (i.) the
other kidney is shown by X-ray to be healthy; and (ii.) there is no tuber-
culous disease elsewhere in the urinary tract, lungs or intestines. It is
important not to wash out the bladder in tuberculous cases. Heliotherapy,
properly carried out, is advisable in some cases, and a course of tuberculin
gives good results.

A **diminution in the specific gravity** *when marked and continuous, even
in the absence of albumen, is suggestive of* CHRONIC INTERSTITIAL NEPHRITIS,
or more rarely DIABETES INSIPIDUS. A **marked increase** *in the specific
gravity is suggestive of* DIABETES MELLITUS.

§ 332. The other **causes of altered specific gravity** are relatively less
important, because they are identified mainly by other means. Never-
theless, the specific gravity of the urine is an extremely important feature,
because, in the absence of sugar, it is a MEASURE OF THE NITROGENOUS
and SALINE EXCRETION, the specific gravity being higher in direct pro-
portion to the amounts contained in a given sample of urine. Therefore,
with certain reservations about to be mentioned, it is a very fair measure
of the FUNCTIONAL ACTIVITY of the secreting substance of the two kidneys
taken together. For example, when one kidney is known to be diseased
or destroyed, it will give us a good idea of the condition of the other,
and in nephritis we may learn something of the amount of renal epithelium
undamaged. In such cases regular estimations of the urea secreted should
be made (§ 306). The reservations just alluded to are four in number:
(1) the specific gravity must always be considered in relation to the total
diurnal quantity of the urine; (2) the total urea varies considerably with
the body weight, being less in women and persons of slight build; (3) it
varies *to some extent* also with the amount of proteid food ingested, and
the work done by the body—thus it is rather less in a person lying in
bed; (4) it is assumed that the liver is healthy because, as mentioned in
the introduction to Chapter XII., the first stage in the manufacture of
urea takes place there, only the concluding stage being performed by the
kidney.

The variations in the *total output of urea* have been mentioned under
the several diseases of the kidney.

The specific gravity is DIMINISHED in—

1. Chronic Interstitial Nephritis.
2. Polyuria, and all the diseases about to be mentioned under that heading, except-
ing Diabetes Mellitus.
3. Myxœdema and other conditions where the nitrogenous disintegration within
the body is diminished.

The specific gravity is INCREASED in—

1. Diabetes Mellitus (owing to the sugar).
2. Some renal diseases where the quantity of water is considerably diminished, such as acute Nephritis or the Cardiac Kidney.
3. Febrile and other conditions where the nitrogenous disintegration is excessive.
4. Whenever the urine becomes concentrated by profuse sweating, vomiting, or diarrhœa.

An increase (POLYURIA), *or diminution, in the quantity of urine is complained of by the patient in several important diseases.*

§ 333. In **Polyuria** it is very desirable to measure the total diurnal quantity, since patients are very apt to mistake increased frequency for increased quantity, and *vice versa.*

There is INCREASED QUANTITY of urine secreted in—

1. *Diabetes mellitus,* which is known by the high specific gravity of the urine and persistent glycosuria.
2. *Diabetes insipidus*—low specific gravity and malaise, but no sugar.
3. *Chronic interstitial nephritis,* which is known by the low specific gravity of the urine, slight albuminuria, etc. (§ 323).
4. *Waxy kidney,* which is known by the low specific gravity of the urine and great albuminuria (§ 324).
5. *Hydronephrosis,* which is known by the passage of large quantities of urine for a limited period of time, accompanied by the disappearance of a swelling from the loin. This is followed by a return to the normal both in quality and quantity of the urine, and then a gradual re-formation of the swelling (§ 341).
6. *Convalescence* after fevers.
7. *Temporary polyuria* occurs in hysteria, nervous excitement, chlorosis, Dietl's crises, alcoholism, and any condition giving rise to a reactionary or paralytic condition of the abdominal sympathetic. Cerebral tumours may be accompanied by polyuria.
8. During the administration of *diuretics.*
9. During the *absorption of exudations,* such as pleural effusion.

There is DIMINISHED QUANTITY of urine in—

1. Acute Nephritis.
2. Subacute and Chronic Tubal Nephritis (some stages).
3. Final stage of Chronic Interstitial Nephritis.
4. The Cardiac Kidney and some other Renal Congestions.
5. Febrile states.
6. Whenever there is profuse vomiting, diarrhœa, or perspiration, or when little fluid is taken.

The patient complains of **polyuria** ; *the urine is of* HIGH SPECIFIC GRAVITY, *and* CONSTANTLY *contains* GLUCOSE (**glycosuria**) ; *there are also fatigue, thirst, and, in spite of a voracious appetite, gradual loss of flesh.* The disease is DIABETES MELLITUS.

§ 334. Temporary Glycosuria may arise in many conditions in which the carbohydrate metabolism is deranged ; often it is of little or no consequence. (1) There may be a temporary diminution of sugar tolerance. (2) Chronic alcoholism. (3) Graves' disease. (4) Pregnancy and suckling (lactosuria). (5) Acromegaly and other conditions, such as tumour, affecting the brain, especially the fourth ventricle. (6) Dietetic errors, as after a heavy meal, especially in the obese. (7) Injury to the liver. (8) Violent emotion. (9) During the paroxysms of ague and collapse of cholera. (10) Chronic Bright's disease and high blood-pressure. (11) Cardiac disease, asthma

and other causes of dyspnœa.   (12) After acute fevers such as influenza or diphtheria.
(13) After much exercise by those unaccustomed to it.   (13) Pancreatic disease.
(14) After epileptic fits.   (15) Renal glycosuria.

Renal glycosuria (Diabetes innocens, renal diabetes). When a small quantity of
sugar is excreted in the urine, and yet the blood sugar is not above normal, the condi-
tion is one of renal glycosuria. The threshold, or point at which sugar is excreted
by the kidney, is lowered. Renal glycosuria may be found accidentally whilst the
urine is being examined. The sugar excretion in this condition is not much affected
by increasing the carbohydrate in the diet; in the true diabetic the contrary is true.
A sugar tolerance test and a study of the blood sugar curve is required before diagnosing
the glycosuria as renal. No treatment is required for this condition.

§ 335. Diabetes Mellitus is a constitutional disease, characterised by
the passage of large quantities of urine containing glucose, associated with
fatigue, progressive emaciation and voracious appetite. 1. There is
usually increased frequency of micturition, and the patient passes large
quantities (6 to 20 pints) of clear pale urine, which has a sweetish odour.
If dropped upon the boot, this leaves a crystalline deposit, by which means
the condition has occasionally been recognised. The specific gravity is
high—1030 to 1040 or more. The amount of sugar varies from 2 to 40
grains or more per ounce, and the total amount per day varies from 10
ounces to 2 pounds. In diabetes the sugar may occasionally disappear
for several days, but in general terms it is permanent and persistent.
The diurnal quantity of urea and phosphates is increased ; diacetic acid
and acetone may be present, and albuminuria sometimes, especially to-
wards the end. 2. Progressive weakness, fatigue and emaciation are
sometimes the first symptoms to attract notice. 3. At other times thirst
or voracious appetite, accompanied by a raw beefy tongue and dry skin,
are the first signs. 4. Complications (vide infra) not infrequently lead
to our detecting the disease, for its earlier stages are often overlooked by
the patient.

Varieties. There are two well-marked varieties of diabetes : (a) The
mild form, which is met with in corpulent middle-aged people, where the
symptoms are moderate, and dietetic restriction removes the sugar from
the urine. (b) The severe variety is met with in acute and chronic forms.
The acute form usually occurs in children or young adults, and occasionally
after head injuries. The chronic form is met with in older people, and
is attributed sometimes to mental worry. It also occurs with other causes
of temporary glycosuria which become chronic.

Causes.—Diabetes occurs in the proportion of three males to two
females. Although it has not always been possible in the past to demon-
strate lesions in the pancreas involving the islets of Langerhans, yet it
is probable that some alteration in structure or function of this special
tissue is the primary, if not the sole cause of diabetes mellitus. The
precise part played in this disease by the liver, which is a storehouse
of glycogen, is still obscure. Three other ductless glands are known to
influence sugar metabolism. (i.) In thyroid disturbance, as in Graves'
disease, glycosuria frequently occurs. (ii.) In hypopituitarism the effect

of a large dose of sugar on the urine and on the blood sugar curve (*i.e.*, a sugar tolerance test) is sometimes studied as a help to diagnosis. The amount of sugar which can be taken without causing glycosuria is greater than normal, *i.e.*, there is increased sugar tolerance. In hyperpituitarism the sugar tolerance is diminished. (iii.) Increase of adrenalin may cause glycosuria. This is seen after an injection or after nervous excitement, as at an insurance medical examination or athletic sports (§ 308).

The *Complications* of diabetes are numerous. In order of frequency . they are : 1. *Phthisis*, which is one of the most frequent causes of death in the condition. 2. Various *skin conditions*, especially eczema, boils, pruritus, and xanthelasma, which appear early in the disease, and carbuncle and gangrene, which appear in the later stages. It is essential to examine the urine for glucose in all cases of boils, carbuncles, and pruritus vulvæ. 3. The *nervous* system is specially apt to be affected, and peripheral neuritis is now known to be frequently caused by diabetes. The knee-jerks are commonly lost in diabetes, sometimes without any other nerve symptom, or there may be tingling, numbness, perforating ulcer, or neuralgia. Restlessness is common, and this may go on to mania or melancholia. The sudden supervention of diabetic coma often terminates life. 4. *Ocular changes* are almost as common as the foregoing, and they may take the form of soft cataract, or defective accommodation leading to a rapidly increasing presbyopia. Retinitis, optic atrophy, and amblyopia also occur.

Diagnosis.—In any of the conditions mentioned under *Complications* the urine should be examined. This is the key to the diagnosis. In *diabetes insipidus, granular kidney, amyloid kidney*, and sometimes in *hysteria* the quantity of urine is excessive, but in none of these conditions is sugar present. Two golden rules will often enable us to identify a case of diabetes which otherwise might be overlooked : Always examine the urine of a patient suffering from (1) boils or from eczema of the genitals ; and (2) apparently causeless wasting.

Prognosis.—1. The glycosuria which is met with chiefly in corpulent persons and others over thirty-five—the so-called " alimentary " glycosuria —has no thirst or other symptoms, but may be true diabetes. Generally with suitable diet the sugar disappears, and the condition warrants an excellent prognosis. 2. In the severer forms the prognosis chiefly turns upon the age of the patient. Before the discovery of insulin if the disease were established in a young adult, life rarely lasted more than two years at the outside. The prognosis is good in cases accompanied by arteriosclerosis, because dieting generally leads to disappearance of the sugar in the urine. Since the discovery of insulin the outlook has much improved, especially in coma. The full result of its use, however, cannot yet be stated. The presence of *complications* other than pneumonia or phthisis does not add very materially to the gravity of the situation. Death may ensue in three ways : (i.) By complications—a third of the cases die of phthisis ; (ii.) asthenia ; and (iii.) with coma, a contributory cause of

which is often a septic focus such as otitis media. Coma is heralded in most cases by certain symptoms which it is well to bear in mind. Such are a decrease in the amount of urine, the occurrence of albuminuria, a rapid increase in the urine of the fatty acid series, viz.: β-oxybutyric acid, diacetic acid, and acetone (§ 313), epigastric pain (often severe), increasing languor, a sighing respiration with extensive abdominal movements (" air-hunger ") and drowsiness. In some cases the coma supervenes suddenly, after a period of excitement. A peculiar sweet odour in the breath, due to acetone, is often a valuable means of diagnosis of diabetic. coma.

Treatment by varying degrees of fasting has been carried out since the days of Naunym. In all cases, when possible, the patient should be confined to bed for the first weeks of treatment. Graham's Ladder Diet is useful to follow:—

For two days only coffee, tea without milk or sugar, meat extract and water are allowed. On the 3rd and 4th days give 5 eggs and 300 grams of green vegetables, cooked with 30 grams of butter. After two vegetable and egg days add 4 ounces of fish. This raises the calorie value to about 920. Two days later, add 2 ounces of bacon at breakfast and omit one egg. The calorie value is now 1195. Two days later, add 2 ounces of sardines at lunch, and omit one egg. The calorie value is now 1335. Two days later, add 2 ounces of ham, and omit another egg. The calorie value is now 1545. Two days later, add 4 ounces of meat, divided into two meals, and omit the fish. The calorie value is now 1745. If the patient is free from sugar when at the top of this " Ladder Diet " add 5 ounces of milk (= 6 gs. of carbohydrate). Two days later, add ½ ounce of bread (= 8 grams of carbohydrate). Increase the bread by the same quantity every other day until carbohydrate tolerance is reached. When carbohydrate tolerance has been reached, as shown by slight return of sugar, give one day of vegetable and egg diet, and then return to the diet on which sugar reappeared, but with only three-quarters of the amount of bread which was then being taken. This is the standard diet for the individual patient. In general terms : give two consecutive vegetable and egg days once a fortnight ; two hunger days, followed by two vegetable and egg days, once a month, returning to the standard diet, as determined for the particular patient, immediately after these days. But the details of the after treatment must depend on the individual case. Variety is achieved by the use of different green vegetables, substitution of sardines and other fish or meat. Some of the butter may be taken on agar biscuits, or cream may be used. Proprietary breads and biscuits are not recommended. When the patient gets up, more food will be needed, but the increase must come from proteins and fats. Alcohol (not sweet wine) is sometimes useful ; the carbohydrate must always be kept low.

The patient is taught to test his own urine and so regulate his own diet ; he learns to live on a diet below his tolerance limit. Fasting should be resorted to in serious cases once a week even when there is no glycosuria ; and all cases do better with a monthly fast. When the carbohydrate tolerance is over 20 grms. give a half-day fast per week. The cases always do best when they are well below normal weight. In severe cases even the vegetables with only 5 per cent. carbohydrate cannot be taken. They must be boiled in three changes of water, which procedure reduces their carbohydrate content by half. Joslin's tables, giving the carbohydrate content in various articles of diet, are reproduced on page 331, and should be constantly referred to when drawing up a régime. With the use of

insulin (an extract of the islets of the pancreas) the value of the low diet is equally great; at the first sign of sugar in the urine, the diet must be reduced. The average dose for the adult is about 10 units daily. Since overdosage by insulin causes too great a fall in the blood sugar, the symptoms of this condition must be carefully watched for by those in charge of a patient under its influence. These may be sweating, weakness, tremors, inco-ordination of movements or of speech, numbness of lips and occasionally diplopia. Such results may be warded off by a hot drink, a tomato or an injection of adrenalin; if these fail a little sugar in water is rapidly effective. For further details on diet and the use of insulin, special textbooks must be consulted.[1]

Many of the advertised starch-free breads are by no means what they claim to be; the careful physician should examine them for starch with the iodine test, and for sugar by boiling them with dilute sulphuric acid, neutralising with caustic potash, and adding Fehling's solution. Saxin is taken in place of sugar.

Insulin has proved of most striking value in the treatment of *coma*. It is safe to give at once 20 to 30 units to an adult in coma. The bowels must be opened and careful examination made for any septic focus, such as boils, otitis media, or pulmonary disease. Saline must be given by a stomach or duodenal tube, but stopped if cyanosis occurs.

The patient complains of polyuria *and many of the other symptoms of Diabetes Mellitus, but the* SPECIFIC GRAVITY OF THE URINE IS LOW, *and there is* NO SUGAR. The disease is DIABETES INSIPIDUS.

§ 336. **Diabetes Insipidus** is characterised by great and persistent increase in the quantity of the urine, without glycosuria and albuminuria, attended by great thirst and emaciation. It is believed to be due to a dilatation of the renal vessels, though how this permanent dilatation occurs is uncertain. The posterior lobe of the pituitary gland helps to control the activity of the kidneys, and indeed is often thought to be responsible for this disease.

Symptoms.—(1) The amount of urine may be very great, from 10 to 20 pints per day. It is pale in colour, so that it may resemble clear water. The specific gravity averages 1002 to 1005. The diurnal amount of solid constituents is as a rule not very much increased, and no other abnormality may be present. Occasionally traces of albumen and sugar appear towards the end. (2) In the mild form of the disease polyuria and thirst are the only symptoms; but in the severer variety nearly all the symptoms mentioned under Diabetes Mellitus are also present—dry skin, emaciation, large appetite, and alternating constipation and diarrhoea. Indeed, it is distinguished from that condition only by the absence of glycosuria. Intercurrent attacks of pyrexia have been observed. (3) Obscure nervous symptoms, with irritability of temper, are common in this disease—such as disturbed sleep, occipital headache, neuralgic pains in the lumbar region, diminished reflexes, and muscular twitchings.

Diagnosis.—The disease is apt in its early stages to be mistaken for *chronic interstitial nephritis*, but the greater age of the patient, the presence of traces of albumen, and of cardio-vascular symptoms, and the absence of thirst and voracious appetite distinguish the latter condition. With *amyloid kidney* there is albumen, and with both *hydronephrosis* and *cystic kidney* a tumour is generally palpable in the region of the kidney. In *Diabetes Mellitus* there is glycosuria.

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[1] *E.g.,* G. Graham, Pathology and Treatment of Diabetes Mellitus.

Causes.—(i.) More males are affected (two or three to one female). Childhood and early middle age are the favourite ages. (ii.) In association with pituitary lesions, injury to the posterior pituitary body, as by tumour, meningitis, trauma, or indirectly by powerful emotions, can be causal factors. (iii.) In one group of cases there is a positive Wassermann.

Prognosis.—The milder varieties may last for a great many years, and exist rather as an inconvenience than as a malady. In the severer forms, especially those due to intracranial tumours, the course may be very rapid, and death ensue in the course of a month. When setting in acutely after injury to the head (which may be attended by some glycosuria at first) recovery may ensue after a year or so. In tuberculous children death usually occurs in the course of one or two years. In general terms, · cases setting in acutely are more hopeful than those which start insidiously. Death may take place from gradual exhaustion, drowsiness passing into coma, with or without convulsions, or from complications such as phthisis or pneumonia.

Treatment.—Most reliance is placed upon hygienic treatment. Tea, coffee, alcohol, and other substances, such as salt, which increases diuresis, should be avoided, but the amount of fluid taken should not be reduced below that excreted. Limit the amount of water which is drunk ; if still there is excess of urine passed, water should be taken freely. Of drugs the favourite used to be valerian gr. v. (0·3) of the powdered root, increased up to ʒ i (4). Pituitary extract, 1 c.c. subcutaneously injected daily, causes diminution of the flow by controlling the vaso-dilatation of the renal vessels. Anti-syphilitic treatment is given when there is a positive Wassermann. Lumbar puncture has been helpful in some cases. When there is disease of the bulb, electricity may be tried—the positive pole placed on the back of the neck, the negative pole passed through the nostril to rest on the cervical spine, ½ to 5 milliamperes for five minutes every second day.

The patient complains that he **cannot pass water,** *and a* DISTENDED BLADDER *can be made out by percussion and palpation above the pubes, or by the passage of a catheter.* The condition is RETENTION OF URINE.

§ 337. The Causes of Retention of Urine come mainly within the province of the surgeon. Those of *sudden* onset are often due to urethral spasm or congestion ; those of *gradual* onset are more numerous. The age and sex of the patient may aid us. Thus, in *childhood* we may suspect impacted calculus, phimosis, or a ligature round the penis ; in *women,* tumours pressing on the neck of the bladder (*e.g.,* fibroid or retroverted uterus), hysteria, or reflex irritation after parturition ; in young or middle-aged *adults,* stricture, gonorrhœa, with congested mucous membrane, spasm after exposure to cold or a drinking bout, or tabes dorsalis ; in *old men,* prostatic enlarge-ment, or atony of the bladder. At all ages there may be calculus or tumour blocking the neck of the bladder, paralysis of the bladder from diseased or injured cord or brain, or reflex spasm after operations about the perinæum.

The *Treatment* is mainly surgical. Before undertaking any operation the blood urea should be estimated. If this is high, over 75 m.g. to 100 c.c. of blood, there is interference with the kidney function and operation is dangerous to life ; drainage improves the condition and operation may be safe later on. In cases of spasm a hot bath or hot fomentations to the abdomen give relief. Hysterical and other nervous affections are referred to elsewhere. Atony and simple vesical paralysis may be treated by nux vomica, and the constant current, one pole being placed on the perinæum and the other just above the pubes.

The patient complains that he has **not passed any water** *for some time, but there are* NO EVIDENCES *of a* DISTENDED BLADDER, *and on passing a catheter it is found to be empty, or nearly so.* The condition is SUPPRESSION OF URINE.

§ **338. Suppression of Urine is** a very grave condition. A catheter should always be passed before the diagnosis of suppression is made. There are two kinds : I. OBSTRUCTIVE suppression, which is due to some obstruction to the flow of urine through the ureters ; and II. NON-OBSTRUC-TIVE suppression, which is due to the non-secretion of urine by the kidneys. The latter form is sometimes spoken of as true suppression.

I. OBSTRUCTIVE SUPPRESSION is due to blocking of both ureters (the kidneys being healthy) by (i.) renal calculus ; (ii.) tumour at the base of the bladder ; (iii.) congenital malformation of the ureters. When only óne ureter is completely blocked, the urine that passes is clear, of low specific gravity, and non-albuminous ; and chronic uræmia ensues until the condition is relieved or the remaining kidney undergoes compensatory hypertrophy (see also Hydronephrosis, § 341). When both ureters are blocked, a condition known as " *latent uræmia* " arises. The *Symptoms* are : the patient passes no urine for about a week, and may complain of nothing except slight drowsiness, but after eight or ten days he becomes restless, with contracted pupils, subnormal temperature, dry brown tongue, and muscular twitchings. In other cases vomiting may be so severe as to suggest the presence of intestinal obstruction. Death is usually sudden, after ten to fourteen days, the mind remaining clear to the end.

II. The causes of NON-OBSTRUCTIVE SUPPRESSION are : (i.) Acute nephritis, or the terminal stage of chronic nephritis (ten to twenty hours befoie death) ; (ii.) collapse (of which suppression is one of the symptoms) —*e.g.*, after abdominal operations or injuries, passage of a catheter, fevers, or local inflammations ; (iii.) hysterical anuria ; (iv.) acute lead, phos-phorus, or turpentine poisoning ; (v.) embolism or thrombosis of both renal arteries (very rare). Whichever of these causes is in operation, the *Symptoms* are : (1) any urine passed is high-coloured and concentrated (high specific gravity), and may contain albumen and casts (indicating that the suppression is due to renal disease) ; (2) there may be urgent vomiting, diarrhœa, and sweating. The other symptoms are those of acute uræmia (§ 296) and those of the cause.

Prognosis.—Suppression is a very serious symptom, though the gravity depends somewhat upon the cause. Of the *obstructive* forms, calculus blocking one ureter, the kidney of the opposite side being healthy, is per-haps the most favourable. If the obstruction affects both ureters and is not removed, death will occur in about eleven days after the obstruction began. In the *non-obstructive* forms death or partial recovery takes place in a few days.

Treatment.—Hót air baths, pilocarpine, and other diaphoretics promote the action of the skin, and so relieve the toxæmia. Free purgation pro-motes the excretion by another channel ; cupping, wet or dry, over the loins relieves the local congestion. In acute non-obstructive suppression good results have been obtained from incision and decapsulation, or both. The kidney is found to be in a state of " cloudy swelling," and when given space to expand recovers its function. As this operation offers the only

hope in many cases, it should be advocated. For the treatment of obstructive suppression a surgeon should be called at once.

The patient complains that his urine dribbles away constantly, *and on percussing over the pubes or passing a catheter, his bladder is found to be empty. He has* TRUE INCONTINENCE. *Or he complains that he has a frequent call to urination, and cannot always hold his water.* He has ACTIVE INCONTINENCE.

§ 339. **Incontinence of Urine** may be of two kinds, and it is best to speak of these as TRUE INCONTINENCE and INCREASED FREQUENCY (Active Incontinence) respectively.

(a) TRUE INCONTINENCE, when the urine dribbles away involuntarily as fast as it is formed, must not be confused with *overflow* or *false incontinence*, which is due to the overflow of a distended bladder in *retention*. The latter is recognised by the percussion signs of a full bladder and by the relief afforded by the passage of a catheter. In true incontinence, which is relatively a rarer condition, the *Cause* is generally quite apparent, such as vesico-vaginal fistula, paralysis and dilatation of the sphincter after the operation of lithotrity, or the paralysis of the sphincter associated with various cerebro-spinal affections.

(b) INCREASED FREQUENCY OF MICTURITION, or, as it is (unfortunately) sometimes called, *Active Incontinence*, is a very common complaint. The patient can hold his water, but the calls to urinate are too frequent, and sometimes so urgent that a few drops dribble away before arrangements can be made. The normal time during which the urine can be retained varies in different individuals, and also according to the amount of fluid taken ; but four or five hours is a fair average. It is longer in the female than the male ; some women can retain the urine for ten or twelve hours. The habit is injurious, and is said to lead to flexions of the uterus.

Increased frequency is due to many *Causes*. The first point to determine is whether there is any marked increase in the diurnal quantity, as in diabetes or chronic granular kidney, because any of the causes of polyuria (§ 333) may be a cause of increased frequency of micturition. In young adults diabetes is perhaps the commonest, but in advancing years granular kidney and enlarged prostate are by far the most common causes. Indeed, our attention is often first drawn to the latter condition because the patient develops a habit of rising at night to pass water. It is not always easy to decide whether the quantity is increased or not, as the patient is apt to think that, because he passes water too often, he passes too much. But having as far as possible excluded polyuria, there remain three groups of causes to consider : 1. Some cause of *local irritation* is undoubtedly the most frequent. The *urine* may be too acid. *Bacilluria* may for long cause no symptom except increased frequency of micturition ; this is a common history in coli bacilluria. The *bladder* may be irritable, as from the presence of an enlarged prostate (the usual cause of abnormal frequency in old age), chronic cystitis, ulceration, tumour, stone (in the young) oxal-

uria, or pressure upon the viscus by a displaced uterus. Or the irritation may be in the *kidneys* from the presence of stone, tubercle, or other cause of pyelitis (§ 331). Or the irritation may be *reflex,* from disease in the vicinity of the bladder, worms, phimosis, or too long a prepuce (a very frequent cause of nocturnal incontinence in children), fissure, piles, prolapse or polypus of the rectum, vascular urethral caruncle (a cause frequently overlooked in women), pelvic inflammation, or varicocele. 2. *Constitutional* causes are occasionally associated with this condition, such as hysteria, sexual excesses, adenoid vegetations in the pharynx, and other causes leading to deficient aeration of the blood. 3. *A congenital* want of development of the sphincter is sometimes present. True congenital cases are rare, and defective action of the sphincter is more frequently due, especially in women and children, to some of the reflex causes above mentioned, the habit persisting after the cause has been removed.

NOCTURNAL INCONTINENCE in children is a troublesome condition often met with in private practice. In such cases we must first satisfy ourselves of the absence of any organic disease. Having done this it is well to remember in this condition that it may be associated with incipient insanity in childhood, general debility, stone in the bladder, and adenoid vegetations in the pharynx. The last named, if severe, result in a deficient aeration of the blood and an unduly heavy sleep. The other causes mentioned above should also be remembered.

Both *Prognosis* and *Treatment* turn almost entirely upon the cause, and are hopeful in proportion as this is removable. The power of retention of the urine is a habit which can be cultivated in early life, and the relative frequency in different individuals varies with habits engendered in childhood. Careful examination should be made to exclude local causes. If the urine is acid, or the bladder irritable, much good may be done by the administration of alkalies and hyoscyamus. If the bladder is wanting in tone, belladonna and nux vomica are the two sovereign remedies. Tincture of rhus aromatica, ♏ v. to ♏ xv. (0·3-1), has been found to be useful where no cause is obvious, and thyroid extract succeeds in certain cases.[1] If there is irritability of the nervous system bromides are specially useful. Children of faulty habits may be treated by sleeping on hard mattresses, or by preventing them sleeping on the back by means of a reel of cotton fixed to the sacrum by plaster. They should be made to pass water before going to bed. Raising the foot of the bed and cold douching to the spine are recommended. Parents should be cautioned against punishing children for this nocturnal incontinence. Electricity correctly applied is of great value.

§ **340**. *The urine presents a* **cloudiness,** *due to some* CRYSTALLINE *or* OTHER DEPOSIT ; it may be URATES, URIC ACID, PHOSPHATES, OXALATES, or FAT, unless it be pus (§ 309), blood (§ 314), or bacteria (§ 313).

[1] Dr. Leonard Williams strongly advocates the use of thyroid extract, gr. ¼ (0·016) *ter die.* cautiously increased, combined if necessary with Calcium Iodide, gr. ii. (0·13), and Liq. Arsenicalis ♏ ii. (0·13). " Adenoids, Nocturnal Enuresis, and the Thyroid Gland " (John Bale and Sons, 1910).

With excess of URATES *the urine,* CLEAR *when first passed, becomes cloudy, with a pinkish* AMORPHOUS DEPOSIT *when it gets cold ; the deposit dissolving again when heated in a tube.* This condition is still by most believed to be due to functional derangement of the liver, and its symptoms are described in the disorders of that organ (§ 274). Various other conditions with which excess of urates and uric acid in the urine may be associated, as a more or less subordinate symptom, have already been referred to in § 317.

The clinical significance of uric acid and urates is still a subject of debate. The deposit may be physiological when occurring after a heavy meal or undue exercise.

In *Multiple Myeloma* the urine is usually cloudy on standing or even passing, due to the presence of the Bence-Jones protein.

Phosphaturia *is usually indicated by cloudiness in a neutral or alkaline urine* (§§ 311 and 317). It indicates decreased acidity of the urine rather than increased excretion of phosphates. (1) Phosphates frequently occur in the urine in such quantity as to cause a turbidity even when *first passed.* They are apt to appear especially towards the end of micturition, not infrequently alarming the patient unnecessarily. Phosphates may be especially abundant in the alkaline " tide " of the early morning or after dinner, and may cause an iridescent " scum " on the surface of the water. There may be no symptoms, even when phosphates are passed in large quantities ; but more frequently phosphaturia is accompanied by chronic dyspepsia. Phosphaturia used to be thought to be due to nerve strain, but it may occur with any cause of (1) wasting or with (2) depression and anxiety, when it is probably due to defective acid formation and lowered metabolism. Phosphates in *excess* occur with (3) hyperchlorhydria, wasting disease and (4) after a diet rich in fruit and vegetable. Phosphates are *diminished* in pregnancy and in convalescence after fevers. A deposit of triple phosphates in freshly passed urine indicates decomposition going on in the bladder.

The *treatment* of phosphaturia is based on the cause. Usually the condition responds to nux vomica with acid tonics (phosphoric or hydrochloric), combined with rest or wise regulation of work and worry. As there is evidence that disorder of the calcium metabolism affects the phosphates, success often follows a diet poor in calcium. Therefore milk, eggs, fish and fruit are cut out and potato and other foods poor in calcium content are given freely.

Oxaluria *is generally indicated by a " powdered wig " deposit on the top of the mucus which settles at the bottom* (§ 317). Transient oxaluria has no clinical significance except as indicating the *nature* of a stone, which has revealed its *presence* by other symptoms. It is also found after a diet of rhubarb, tomatoes, sorrel, strawberries, spinach, tea and coffee, or cocoa. But oxaluria is also connected with other clinical conditions. (1) Excessive formation of oxalates in the urine. Cases have been recorded where the symptoms of rapid emaciation and pains in the loins and back were attended by an excess of oxalates in the urine. (2) Pancreatic disease. (3) Other observers have connected certain nervous symptoms, such as mental depression ; it is probable that these symptoms are connected with the concurrent dyspepsia and pains. (4) Oxaluria is associated with abnormal fermentation of sugar. Urates are generally precipitated in the urine at the same time as the oxalates. (5) Oxalates are found in large excess in paroxysmal hæmoglobinuria (§ 328) and their presence may cause hæmaturia and albuminuria.

Treatment consists in avoiding the foods above mentioned which contain oxalates. A diet of milk, peas, eggs, meat, oats, rice, butter, pears, melon, and grapes is recommended. Lemon juice is valuable, but excess of sugar must be avoided. The formation of crystals is prevented by the ingestion of magnesia and citrate of potassium. Calculi of oxalates are reduced by rendering the urine acid with acid sodium phosphate.

Fat may occur in the urine in chronic tubal nephritis attended by much fatty degeneration of the epithelium, and after fractures of the bones. It is found in great abundance in **Chyluria**. The presence of chyle in the urine gives to it a milky white appearance and the power of coagulating. Chyluria is not uncommon in the tropics, where it is due to the migration of the filaria sanguinis hominis from the lacteals into the urinary tract, the unnatural communications thus made leading to the paroxysmal appearance of chyle in the urine. The urine passed at night is the more completely white; that passed by day may be mixed with blood. The embryos of this parasite are to be found in the urine with a few red and white blood-cells, albumen, fat, and shreds of fibrin. Chyluria may follow trauma, and it may accompany leukæmia in rare cases.

Prognosis.—The patient may live twenty years with but little impairment of health. In other cases, however, great debility and mental depression may be present.

Treatment.—Prevent the disease by boiling the drinking-water. To meet the drain on the system give plenty of nourishing food.

In *Pseudo-Chyluria* the milky appearance of the urine is due to the presence of the same material that occurs in pseudo-chylous ascites.

§ 341. **Renal Tumours** may be of six kinds: (I.) HYDRONEPHROSIS; (II.) PYONEPHROSIS; (III.) PERINEPHRIC ABSCESS; (IV.) MALIGNANT DISEASE; (V.) CYSTIC DISEASE; and (VI.) MOVABLE KIDNEY. The last-named is described under Abdominal Pain (§ 201), which is the symptom for which advice is sought. Extravasation of blood after injury to the kidney may simulate a tumour (§ 327).

The *Physical Signs* common to all tumours of the kidney, and their diagnosis from other ABDOMINAL TUMOURS, are given in §§ 212 and 318.

I. **Hydronephrosis** is a term indicating a cystic tumour of the kidney, caused by the gradual obstruction of the urinary passages, and the consequent dilatation of the pelvis of the kidney.

The *Symptoms* by which this tumour is recognised are: (1) At intervals a large amount of urine passes, with concomitant reduction or even disappearance of the tumour. The urine is pale, clear, and of normal composition. (2) Constitutional and general symptoms may be absent. (3) Local pressure symptoms may arise, causing pain or disturbance of function of the neighbouring organs.

Etiology.—The causes of obstruction to the outflow of the urine may be (i.) *congenital* (contracted or twisted ureters); (ii.) *acquired* causes, which may occur (a) in the *urethra*, such as stricture or enlarged prostate.; (b) in the *ureter*, such as occur from stone or blood-clot; pressure by pelvic or other tumours; contraction after operation, injury, or disease of the ureter; or kinking, as in movable kidney. These acquired causes give rise to a gradual obstruction, and when the obstruction is intermittent the tumour may become very large, when it is liable to be mistaken for an ovarian cyst, or even for ascites. In such cases a trocar may be introduced, and the fluid withdrawn would reveal an absence of the albumen which is always present in an ascitic fluid.

Prognosis.—If the condition is unilateral and intermittent it may cause little trouble, and may disappear after a duration of years. On the other hand, a double hydronephrosis is very serious, as it leads to uræmia. The surgeon should be called in early. The complications are rupture

into the peritoneum or pleura ; the onset of suppuration in the pelvis of the kidney (pyonephrosis) ; or uræmia, due to atrophy of the substance of both kidneys.

Treatment.--If the tumour is intermittent, unilateral, and causing few symptoms, it is best to leave it alone. Osler recommends the use of a pad to retain the organ in place and prevent further dilatation. If the tumour becomes very large, surgical treatment is advisable. In all cases the cause must be ascertained and, if possible, treated.

II. **Pyonephrosis** is a cystic tumour of the kidney due to distension of the pelvis and calices by fluid containing pus. It is consequent on obstruction to the free outlet of the urine in septic cases of pyelitis, or sepsis supervening on hydronephrosis.

The *Symptoms* are : (1) The tumour is tender to palpation ; (2) symptoms of pyelitis are present—pyuria, intermittent pyrexia, sometimes rigors, and dull pain in the loin ; (3) at intervals, when the obstruction is removed or diminished, the tumour may subside, coincident with the passage of a large quantity of pus in the urine.

The *Causes* are : (1) *pyelitis* (§ 331), with blocking, partial or complete, of the ureter ; or (2) *hydronephrosis* (*vide* Causes of this above) becoming septic —*e.g.*, from extension upwards of cystitis.

Diagnosis.—(1) From *hydronephrosis*, which has no tenderness or fever ; (2) from *perinephric abscess*, which has greater tenderness in the loin and a more superficial swelling, with local signs of abscess sooner or later.

Prognosis.—The condition is very grave. A tuberculous pyonephrosis may undergo cure by fibrosis ; but in most cases the patient becomes worn out with long discharge, or develops amyloid disease, or a fatal issue is rapidly brought about by the tumour bursting into the abdomen or chest.

Treatment is mainly surgical, and nephrotomy is indicated. The cause must be treated medically.

III. **Perinephric Abscess** is not very uncommon. It may arise by (i.) extension from kidney disease (pyelitis) ; (ii.) extension from a perityphlitic abscess ; (iii.) extension from other organs—*e.g.*, abscess of the liver, empyema or spinal caries ; (iv.) after an injury. The *Symptoms* are : (1) dull, aching pain in the loin radiating down the leg ; (2) deep-seated resistance in the hypochondrium in front, tender to pressure ; (3) the temperature is continuous, or pyæmic in acute cases with sudden onset, or intermittent in insidious cases ; (4) the leg on the same side is kept flexed and the patient stoops when walking ; (5) swelling, with œdema of the skin, which appears late in the disorder, is felt between the iliac crest and the last rib, and it may be fluctuant ; (6) the urine may or may not be altered according to the cause, but traces of albumen are common. The *Diagnosis* is difficult in the early stage when pain alone is present, when it may readily be mistaken for *lumbago* or *spinal disease*, but there is no fever in the former. Later it may be mistaken for a *renal tumour*, but in a simple tumour fever is absent, and the leg would not be held constantly flexed ; the aspirating needle may be used. In *pyonephrosis* there is not such acute pain or tenderness. *Prognosis.*—The abscess tends to open or to burrow its way in various directions, into the alimentary or urinary canals, peritoneum, or pleura. It may point in the lumbar region or various other directions, and burrow for a considerable distance. *Treatment.*—In the early stages, before the diagnosis can be certain, give hot fomentations and opium for the pain ; as soon as pus is recognised operative procedure is necessary.

IV. **Malignant Disease starting in the Kidney** is certainly a rare condition, as it has only been found in about 1 in 500 autopsies on persons dying of malignant disease.[1] It affects children under nine (in whom *sarcoma* chiefly occurs), and adults over forty (in whom usually it is *carcinoma*), there being a remarkable immunity be-

[1] Discussion on Renal Tumours, Path. Sec. B.M.A., 1899.

tween these age periods.[1] Renal sarcoma is the commonest abdominal growth in children, and it is believed often to start before birth. According to Bland-Sutton, it is met with in the first five years of life, and then, after a period of immunity, is found again in people between fifty and sixty.

The *Symptoms* are : (1) The tumour is rapidly growing, usually of firm consistence, but if of very rapid growth it may appear fluctuating ; (2) hæmaturia, frequent, intermittent, and of moderate amount ; (3) progressive emaciation ; (4) the pain is variable, sometimes it is very severe, owing to pressure upon or infiltration of the neighbouring organs. Sometimes pain is entirely absent, and the tumour may have attained a very large size before any symptoms occur.

Diagnosis.—When a tumour occurs in a movable kidney it is apt to be mistaken for *ovarian tumour* or *fibroid*, and vaginal examination is necessary (see § 212 for diagnostic points). *Tuberculous* kidney in a child may present difficulty, but the pain is less, and pyuria is present rather than hæmaturia. *Pyonephrosis* is accompanied by fever, the swelling is fluctuant, and there is a history of pyuria. Retroperitoneal and renal sarcoma are the chief causes of enormous abdominal tumours in children. The diagnosis of malignant tumours is not usually difficult.

The *Prognosis* is very grave. If untreated, death occurs in six to twelve months after detection of the growth, the cancer of adults being of somewhat slower growth.

Treatment is usually too late ; early excision gives the only chance of life.

V. Cystic Disease of the Kidneys is a rare condition, usually of congenital origin, in which both kidneys contain cysts of varying size and number.

Varieties.—(i.) The cystic kidney in its typical form is a mass of cysts, and is usually congenital ; (ii.) cystic kidney may arise in connection with granular kidney ; in this variety the tumour is never so large as in the former ; (iii.) cystic formations may also be due to hydatid.

Symptoms.—(1) There is a swelling usually in both lumbar regions, of insidious growth, very hard at first, and later yielding. (2) The other symptoms are similar to those of chronic interstitial nephritis—the urine is abundant, pale, of low specific gravity, containing traces of albumen, and occasionally blood and casts. The heart becomes hypertrophied, and the pulse indicates high blood-pressure. The patient may have excellent health for many years, or may develop symptoms of chronic uræmia.

The *Diagnosis* may be difficult. When symptoms of granular kidney occur, together with a tumour in both renal regions, the condition may be diagnosed as Cystic Kidney. The tumours have to be diagnosed from other abdominal tumours (§ 212).

Causes.—The disease may occur in the fœtus. Patients with the above symptoms, however, are usually men over middle age. Out of twenty-one cases collected by Dr. W. H. Dickinson, eleven were over forty. •

Treatment is similar to that of nephritis. Death may occur from uræmia or the same complications as those of interstitial nephritis.

Hydatid cyst may occur in the kidney, and may be difficult to differentiate from other cysts unless it opens into the pelvis of the organ, when the characteristic hooklets (§ 279) are found in the urine. The passage of vesicles may cause renal colic. The condition may be suspected if (i.) the tumour has the " hydatid thrill " on palpation ; (ii.) there is evidence of the presence of cysts elsewhere ; and (iii.) there is a history of residence in affected countries. (iv.) Eosinophilia is present.

The *Prognosis* is not grave. The cyst may last for years with symptoms, or it may burst into the pelvis of the kidney. It may open into the stomach or bowel, with ulterior recovery ; or into the chest, which is a serious complication. It may become very large and give rise to pressure signs.

Treatment is surgical.

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[1] The solid tumours affecting the kidney consist of (*A*) *Connective tissue type* :— I. Simple or benign growths (fibroma, lipoma, angioma) ; II. Sarcoma, which is by far the commonest. (*B*) Growths of an *epithelial type* :—I. Adenomatous growths (simple adenoma, trabecular, and papilliform cystomata) ; II. True Carcinoma :—(1) glandular type ; (2) malignant papilloma. (*C*) Adrenal inclusions. (*D*) Adrenal growths.

CHAPTER XIV

DISEASES PECULIAR TO WOMEN

THE symptoms and consequences which may arise from disorders of the female genito-urinary organs are very numerous and widespread. Indeed, there is hardly a physiological system which does not suffer when these organs become affected. It is on this account that they should receive more attention from the general physician than is the custom.

PART A. SYMPTOMATOLOGY

§ 342. The symptoms proper to these organs may be divided into *local* and *general*. The LOCAL SYMPTOMS are certain external conditions around the vaginal orifice, leucorrhœa (vaginal discharge), dysmenorrhœa (painful menstruation), menorrhagia (excessive menstruation), amenorrhœa (deficient menstruation), pain in and around the organs, various disorders of function (*e.g.*, dyspareunia and dysuria) and tumours of the uterus.

The GENERAL SYMPTOMS consist of (1) malaise and general ill-health, which is often quite out of proportion to the amount of local mischief. A life of chronic invalidism not infrequently supervenes upon some chronic though slight derangement of the reproductive organs. This general weakness is specially apt to affect the nervous system, and one is sometimes tempted to credit the older authors who named hysteria on account of its supposed origin in the womb (ὕστερος.) (2) " Dyspeptic " symptoms of a reflex kind are nearly always present, as in other disorders connected with the abdominal viscera. (3) Anæmia is another consequence, though this may be due in part to the confinement indoors, or to the " loss " in cases of excessive menstrual flow. (4) Various neuralgiæ and a general hypersensitiveness of the sensory and sensitive apparatus. A certain degree of this is normal during the menstrual periods, and as civilisation advances it seems as though this recurrent hypersensitiveness were increasing. By degrees, especially in those who suffer from dysmenorrhœa, this undue generalised hyperæsthesia is prolonged into the intervals between the periods.

Case-taking in diseases of women differs somewhat from that given in Chapter I. The following summary will form a guide to the principal questions to be answered as a matter of routine :

1. What is the leading symptom complained of by the patient ?

2. History—name, age, married or single. (a) If married, how long? How many children? Date of last confinement? Any miscarriages? Confinements easy or difficult? How long in bed after the birth?

(b) Menstruation—age at which it commenced? (i.) Regular? Twenty-eight day or thirty-day type? Lasting—three, five, seven days? (ii.) Blood coming in clots (means excess)? (iii.) Painful or not? Pain dated back to a particular time? Pain in small of back, shooting down one or both legs, or in ovarian region? Pain persistent or paroxysmal? What relation to the flow?

(c) Any intermenstrual discharge—duration; quantity; white, clear, or thick and yellow; offensive; or with débris and blood.

(d) Micturition—painful, dribbling, or too frequent. Condition of bowels—pain on defæcation?

(e) Other physiological systems to be inquired into; and whether general health has suffered.

PART B. PHYSICAL EXAMINATION

§ 343. Except in certain circumstances, an abdominal and local examination should be a matter of routine in all gynæcological cases which are not on the surface obvious. There are four methods by which the female pelvic organs can be investigated.

(a) An EXTERNAL EXAMINATION of the abdomen - inspection, palpation, percussion, auscultation (§ 190).

(b) A VULVO-VAGINAL EXAMINATION should not be undertaken without duly considering both the necessities of the case, and the feelings of the patient. The patient should lie on the back, with the legs both drawn up at an acute angle; the light should come from the foot of the couch. Note by inspection the colour and condition of the vulva, hymen, urethral orifice, and the condition of the perineum, especially in women who have borne children, and then proceed to pass the finger gently. Some use the first, others the second finger; it is useful to be able to use either hand, so that we may keep one hand for possibly septic cases alone. The finger-nails should be kept extremely short and smooth, both for the patient's comfort and for cleanliness.[1] As a lubricant for the finger some physicians like sanitas with vaseline (about 5 per cent.), others use carbolised glycerine (1 in 200). When there is much vaginal discharge rubber gloves should be worn lubricated with glycerine. The finger is passed well in, and the condition of the vaginal walls noted; the position and condition of the cervix, whether patulous and soft as in pregnancy, firm, granular, fissured, conical, etc. Note also any fixity of the uterus, and whether there is an angle or dip between the cervix and body anteriorly or posteriorly such as occurs in flexions.

(c) It is very desirable to make a BIMANUAL EXAMINATION next in order. Instruct the patient to lie on her back, to draw up the legs, and

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[1] A story is told of the late Mr. Lawson Tait, whom a great many foreigners used to visit. One particularly insistent gentleman, who generally had long dirty nails, was always seeking to ascertain from him the secret of his success. Lawson Tait, who was not in the habit of measuring words, became somewhat annoyed at the insistence of his visitor, and one day in reply to the oft-repeated question, he said: "The secret of my success is that I keep my nails short and extremely clean."

relax the abdominal muscles. With the finger of the right hand in the vagina, the physician places the left hand firmly above the brim of the pelvis, so as to be able to manipulate the uterus between the two hands. Note the size, position, and mobility of the uterus, the presence or absence of tumours, displacements of the uterus, or pelvic swellings or exudations. The bladder must be empty, and the rectum if possible.

(*d*) Various INSTRUMENTS are of considerable aid.

1. The *Sound* must be used but rarely, and only with strict antiseptic precautions. Undoubtedly harm used to be done by passing it through a septic vagina into the uterus. Its use is contra-indicated in (i.) pregnancy, (ii.) menstruation, (iii.) acute inflammation in the pelvis, (iv.) cancer, and (v.) it should never be passed before making a bimanual examination.

With the tip of the right forefinger against the os pass the sound along the palm of the right hand until it slides well into the cervix. Then by a gentle turn and by a very gentle pressure upwards the sound will pass upwards and forwards into the uterine cavity.

The uses of the sound are to discover : (1) the depth of the uterus, which is normally 2½ inches, and the thickness of its wall ; (2) the position of the uterine cavity, when it is impossible to find it by bimanual examination ; (3) the state of the endometrium ; (4) the size of the os ; (5) the presence of tumours in the uterus.

2. *Vaginal Speculum.*—Many different specula are in use. Practically they are of three types. The Ferguson, which is a tube ; the bivalve or trivalve, which consists of two or three limbs jointed together ; and the duckbill, which consists of two separate pieces. The first is best for the examination of the os ; the second for the examination of the walls of the vagina ; and the third for operative measures. In passing it do not forget the vaginal canal is directed backwards and upwards, and less pain is produced by quick movements in the right direction than by slow bungling. Note the condition of the mucous membrane, and the character of any discharge. If it be desirable to make some application to the interior by means of a Playfair's probe, this should be done before withdrawing the speculum.

3. The *volsellum* is a hook for drawing down one or other lip of the cervix, which is desirable, for example, (i.) for the introduction of tents. It is also of use to examine (ii.) any catarrhal patch, (iii.) where the uterus is freely movable, and (iv.) to palpate the posterior surface of the uterus. It is contra-indicated in those conditions in which the sound is contra-indicated, and also in tubal pregnancy.

DILATATION OF THE CERVIX may be done by two methods :

1. *Slow Method.*—Sea tangle, tupelo, or sponge tents are inserted into the os uteri, and left *in situ* for some hours. By the absorption of fluid they swell up and distend the cervical canal. This method is useful in nulliparous women or when the cervix is rigid. It is little used nowadays.

2. *Rapid Method.*—Hegar's or Fenton's dilators are usually employed. They are vulcanite or metal instruments of graduated sizes. General anæsthesia is necessary. Having inserted the posterior vaginal speculum, fix the anterior lips of the cervix with the volsellum or ovum forceps, draw well down, and insert the dilators gradually one after the other until the cervix is large enough to examine the interior with the finger. In this way one can curette the interior if there is any granular endometritis, or make a digital examination of the endometrium, which is possible only after much dilatation. The nature of any growth present is discovered by a microscopic examination of the scraping ; such examination should never be omitted. Dilatation of the cervix is contra-indicated in tubal disease, possible pregnancy, or cancer of the cervix. It should be performed with great caution when the tissues are softened by recent pregnancy.

PART C. DISEASES OF WOMEN, THEIR DIAGNOSIS, PROGNOSIS,
AND TREATMENT

§ **344. Routine Procedure and Classification.**—Having ascertained the
patient's principal or *Leading Symptom*, and the leading facts as to the
History, according to the scheme given in Part B., proceed, unless the
nature of the case is not already apparent, to the *Physical Examination*
(subject to the reservations mentioned in Part B.).

CLASSIFICATION.—The diseases of the female reproductive organs may
· be arranged, like urinary disorders, under the various cardinal symptoms
to which they give rise—viz. :

    (*a*) Morbid alterations of the vulva and external parts .. § 345
    (*b*) Leucorrhœa .. .. § 346
    (*c*) Dysmenorrhœa .. .. § 347
    (*d*) Hæmorrhage .. .. § 348
    (*e*) Amenorrhœa .. .. § 355
    (*f*) Pelvic pain, acute (§ 358), chronic .. .. § 362
    (*g*) Pelvic tumours .. .. § 363
    (*h*) Pain on sitting, dispareunia, dysuria, and other disorders of
        function .. .. § 367

§ **345. Morbid Alterations of the Vulva.**—A few of the common altera-
tions are enumerated here.

VULVITIS in children may be caused by the migration of round worms,
by uncleanliness, debility, gonorrhœa, or bad habits. In adults it is
generally accompanied by vaginitis (*q.v.*).

PRURITUS VULVÆ (itching) is sometimes a very troublesome condition.
An examination should always be made to discover whether eczema,
pediculi, or irritating discharges be present. If these be absent diabetes
may be suspected.

ECZEMA of the vulva is, in the author's experience, greatly on the
increase, as a consequence, in his belief, of the modern fashion of wearing
closed non-washable knickers.

CARUNCLE is a minute red irritable papilloma situated usually just
within the urethral orifice. It is a frequent cause of painful micturition,
painful sitting, and painful coitus. There is also a painless form.

Slight prolapse of the urethra may give rise to a red swelling which may
be mistaken for a caruncle.

LABIAL THROMBOSIS is readily recognised, and is a not infrequent condition in
certain hyperinotic states.

ABSCESS of the vulva sometimes follows the last named. Sometimes it occurs as
an inflammation of Bartholin's gland.

HERPES is an eruption of a small group of vesicles. They readily rupture, leaving
round superficial ulcers which may become infected secondarily.

NOMA, DIPHTHERIA, CHANCRES, CONDYLOMATA, ULCERS (simple or malignant)
also affect the part.

In the *Treatment* of vulval conditions cleanliness is essential, and on
· the whole the lack of this is one of the most frequent causes of vulvitis.
It is surprising what little attention is paid to this matter, as is shown by

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the immense quantities of epithelial cells which are habitually found in the urine. Any eczematous or local condition must be treated as elsewhere. Caruncle is best treated by strong nitric acid or Paquelin's cautery. Labial thrombosis requires surgical treatment. Pruritus vulvæ may in my experience often be cured by large doses of calcium chloride or by local applications of the high frequency current. Cases which have long resisted other treatment have yielded to this. Locally, lotio calaminæ co. in weak carbolic acid solution, liq. carbonis detergens, and sodium bicarbonate and borax solution are employed in varying conditions. In cases where the itching is very intense, a solution of nitrate of silver (4 per cent.) may be painted on, the parts having been first anæsthetised by the application of cocaine solution. At the same time use internal remedies, such as arsenic, quinine, hexamin, and bitter tonics. For herpetic ulcers use 5 per cent. nitrate of silver, and then zinc oxide paste.

§ 346. **Leucorrhœa** is any white or whitish discharge from the vulval orifice (colloquially known as the " whites "), due to excessive secretion from the mucous lining of the genital tract. It may be caused by an unhealthy condition of the mucous membrane of the Fallopian tubes, or of the body or cervix of the uterus, or of the vagina. For a correct diagnosis of the cause it is necessary to make a careful investigation of the discharge.

(*A*) LEUCORRHŒA OF VAGINAL ORIGIN arises when there is vaginitis from any cause, either acute or chronic.

(*a*) In ACUTE VAGINITIS the discharge is profuse, yellow or greenish, and sometimes blood-stained, attended by dysuria and local signs of inflammation. The chief *Causes* of acute vaginitis are : (1) Traumatism, due to pins, peas, and worms in children, or in the adult an irritant pessary, or other foreign body (contraceptive appliances, etc.), too powerful injections, or excessive coitus ; (2) gonorrhœa, which is hard to diagnose from non-specific acute vaginitis except by the microscopic examination of the discharge ; (3) spread from adjacent parts, and (4) a diphtheritic form. A severe acute vaginitis is probably of gonorrhœal origin, and the danger of this rests in the liability to endometritis, pyosalpinx, peri- or parametritis, cystitis, and ascending pyelitis. The B. coli, streptococci, diplococci of various kinds, fungi, and pyogenic infections, also cause acute vaginitis. *Treatment* consist of rest, saline purges with hyoscyamus to allay the pain, copious warm drinks, hot hip-baths, and douches of carbolic (1 or 2 per cent.), potassium permanganate (10 grains to the pint), or corrosive sublimate, and after a few days some astringent lotion such as sulphocarbolate of zinc ℨ ïi–O ï (8–600), glycerine of subacetate of lead ℥ss–Oi (16–600). Tr. Iodine (ℨ ï–O ï), Jeyes' Creolin (ℨ ï–O ï), protargol (4 per cent.) may be applied through the speculum. Iodoform or protargol pencils may be inserted daily.

(*β*) In CHRONIC VAGINITIS there is a thick, continuous, opaque discharge, with or without local signs of inflammation, according to the cause in operation. The *Causes* are (1) antecedent acute vaginitis ;

(2) various constitution conditions, such a general debility, strumous (*i.e.*, tuberculous) diathesis, diabetes, old age, alcoholism, anæmia, syphilis, rheumatism, and convalescence from fevers; (3) new growths in the vaginal walls, such as epithelioma; (4) irritant foreign bodies and other causes mentioned under Acute Vaginitis. The *Treatment* consists of the appropriate remedies for any constitutional disease present, combined with warm douches (100° F.), containing sulphate of zinc or sulphate of copper, or the remedies mentioned under Acute Vaginitis. Local applica- ·tions are made with Ferguson's speculum, and a cotton swab dipped in 2 per cent. iodine solution or silver nitrate solution (5 per cent.). Medi- cated pessaries may be used at night.

(B) LEUCORRHŒA OF UTERINE ORIGIN may be due to endocervicitis or endometritis, cancer of the uterus (see Hæmorrhage), salpingitis, concurrent peri- or para-metritis (see Pelvic Pain)—and, lastly, to constitutional causes such as gout, rheumatism, or anæmia.

I. In ENDOCERVICITIS (CERVICAL ENDOMETRITIS), or inflammation of the cervix, the discharge is more or less constant, and usually consists of *glairy material* like white of egg, but it may be muco-purulent. The other symptoms are : (1) The cervix is swollen, and may present retention cysts, but more usually on examination with the speculum one sees an " erosion " or catarrhal patch, which may bleed slightly on pressure ; (2) menorrhagia or dysmenorrhœa and backache are frequently present. Endocervicitis may have to be *diagnosed* from *cancer* of the cervix. Here the age is not much guide, as cancer of the cervix may come on in a patient as young as twenty-six. Cancer is hard to the touch and is friable, readily breaking down and bleeding when touched, and there is usually a blood-stained discharge. Microscopic examination of scrapings will determine the diag- nosis. When fixity of the uterus and cachexia have appeared, the diagnosis is simple. For *Causes* and *Treatment* see below.

II. In ENDOMETRITIS, or inflammation of the lining membrane of the body of the uterus, the discharge comes in gushes when the patient rises or walks about ; and in the senile it may be blood-stained. Endometritis is usually accompanied by both menorrhagia and dysmenorrhœa, and general pelvic discomfort and pain. The general health may be poor. Bimanually, the uterus is found to be enlarged ; the cervix is often hyper- trophied and inflamed. Sometimes there is a history of recurring abortions or of sterility. Endometritis may require to be diagnosed from cancer. Owing to the risk of delay, curettage should be performed, and the scraping thus obtained determines the diagnosis.

The *Causes* of endocervicitis and endometritis are classified thus : (1) Bacterial invasion—gonorrhœal, diphtheroid, coliform, septic, and other infections, spreading upwards ; or from retained products after labour or abortion, or the use of dirty instruments ; (2) congestion of the uterus, as in displacements, tumours, injury, subinvolution, tumours · of the adnexa, excessive coitus, constipation ; cardiac, pulmonary, and renal disease ; old age.

Treatment.—Endometritis and endocervicitis require first a certain amount of hygienic and general treatment, especially if there has been much menorrhagia. In mild cases, tepid douches (1 quart) of alum or chloride or sulphate of zinc, ʒ i–O i, may be enough. Displacements and other causes of congestion must be rectified. The special organism responsible for any infection must be treated. For endometritis resisting such treatment there are three methods of local treatment : (1) The application of strong carbolic, protargol, or other corrosive to the interior by means of Playfair's probes ; (2) dilatation of the cervix and curetting the interior ; (3) ionisation. Endocervicitis is treated (after removing mucus with 25 per cent. liq. potassæ) with a saturated solution of picric acid on wool wrapped round a Playfair's probe, and passed through a Ferguson speculum to protect the vaginal wall. Ionisation and diathermy give excellent results in both endometritis and cervicitis. Operative treatment is only rarely necessary.

§ 347. Dysmenorrhœa is pain during the menstrual period. There are three varieties : (I.) NEURALGIC or SPASMODIC, in which the pain is paroxysmal, and may be so severe as to cause vomiting and collapse. It is situated chiefly in the hypogastrium, begins a few hours before the flow, and lasts for one or two days. (II.) In the INFLAMMATORY form the pain is dull, aching, persistent, situated sometimes in the small of the back, and down the legs, begins several days before the flow and is relieved by the flow, especially when it is profuse. (III.) In the MEMBRANOUS variety the pain is severe, paroxysmal, and relieved as soon as the membrane is passed. In the *first* named, local examination reveals nothing wrong in the uterus or its appendages, and the menstrual flow is usually natural. In the *second* variety, examination generally reveals some abnormality in the uterus or its appendages—*e.g.*, endometritis, fibroids, adhesive bands, ovaritis ; and it not infrequently dates from a confinement or abortion. The *third* is diagnosed by the passage of a membrane, and is distinguished from abortion in that it is passed every month.

Causes.— Spasmodic dysmenorrhœa dates from the onset of menstrual life and is probably due to imperfect muscular development and to various causes of neuralgia. The causes of varieties II. and III. are such as lead to inflammation of the uterus or its appendages. All three varieties have been variously ascribed to obstruction of the flow by flexions of the uterus or by constriction of the cervix, or to the undue excitation of uterine contractions.

Treatment.-- I. The neuralgic form usually calls for general treatment —hygienic, dietetic, and tonic. To encourage free circulation, exercise should be taken before and between the periods. Treatment directed to the diathesis, as in rheumatic persons (aspirin, guaiacum resin, gr. x. (0·6)), may effect a cure. It is very important in this and in the other varieties to avoid constipation. Warm baths, and especially Turkish baths, are very valuable in my experience. Remedial treatment at the time of the period consists of hot bottles to the hypogastrium, hot drinks, feet in hot water, cannabis indica, liquor sedans, ergo-apiol, belladonna, camphor, sal volatile, bromides, castoreum, antipyrin, essence of peppermint, 5 drops in warm water, and morphia (with great caution). In obstinate cases dilatation of the cervix, applications of the galvanic cur-

rent or diathermy usually cure. Childbirth usually cures the condition.

II. The inflammatory or congestive form admits of the same symptomatic treatment as the foregoing. The remedial treatment should be directed to the lesion which is the causal agent. Depletory methods, such as a glycerine tampon, leeches, or scarification of the cervix, combined with a saline purge, are indicated. Antipyrin is of little use ; alcohol increases the pain. Styptol and pituitary extract cure some cases. Very hot douches (110° F. for ten minutes) should be given twice daily. In severe cases removal of the uterus without the ovaries has been adopted.

III. For the membranous form the symptomatic treatment is as above, with the subsequent dilatation and curetting of the interior during the interval.

Mittelschmerz (middle pain) is a rare condition in which pain is felt at regular intervals between the menstrual periods. It is not so severe as spasmodic dysmenorrhœa. Its cause is unknown.

§ 348. Hæmorrhage.—*Menorrhagia* indicates an excessive flow at the monthly period ; *Metrorrhagia* indicates irregular hæmorrhage from the uterus, irrespective of the period. It is difficult to separate these two symptoms, as their causes are more or less identical, and they very often occur together. Hæmorrhage from the vulva or vagina is usually slight in quantity, and its cause readily discovered by inspection. Hæmorrhage from the cervix is usually due to polypi, malignant disease, tuberculous or syphilitic ulceration. Rarely it is due to erosion or injury by a pessary. All of these are made out on inspection. Hæmorrhage after coitus is suggestive of malignant disease or a polypus hanging from the cervix.

Hæmorrhage from the uterus may be due to the following causes : Endometritis, fibrosis or metritis, the acute fevers, purpura or other constitutional conditions, fibroids, and polypi of the uterus, pelvic inflammations, subinvolution of the uterus, congestion consequent on cardiac or lung disease, malignant disease, retroverted uterus incarcerated in Douglas' pouch, ovarian tumours (occasionally), inversion of the uterus, and extra-uterine fœtation. Flexions and versions of the uterus rarely cause symptoms unless attended by pelvic inflammation or adhesions. A single excessive hæmorrhage may be due to strong emotion, over-exertion or sudden change of temperature.

In women *over thirty-five* the above causes also may give rise to hæmorrhage, but in addition it may be due to the Menopause. The sudden supervention of *metrorrhagia* with *acute pain* should always suggest a miscarriage or an extra-uterine fœtation (§ 355). In women *past the menopause* some gross lesion of the uterus, especially Cancer or Uterine Fibroid, is nearly always present.

Many of these conditions are dealt with elsewhere, but menorrhagia or metrorrhagia is the chief symptom referable to the reproductive organs in : (I.) Certain Constitutional conditions ; (II.) Uterine Fibroid or Polypus ; (III.) Subinvolution (in persons under thirty) ; (IV.) Metritis ;

(V.) the Menopause ; and (VI.) Malignant Disease (in persons over thirty). These conditions will therefore be differentiated here.

§ 349. Hæmorrhage may depend upon certain CONSTITUTIONAL CONDITIONS. (1) Certain women of a plethoric habit of body, usually with florid countenances, may be troubled with too profuse periods all their lives, and a tendency to excessive flow on any trivial exciting cause. (2) Prolonged lactation or too many and too frequent pregnancies ; (3) residence in tropical climates ; (4) acute specific fevers ; (5) mental overwork, especially if combined with a sedentary life, are said to produce it. (6) The vague condition we call hysteria, especially in that variety which is subject to flush storms, is frequently attended by menorrhagia. (7) The congestion in the circulation which attends some heart and liver diseases finds more or less relief in this way. (8) Menstruation may be very profuse at the onset of the function of puberty. The differential characters of the bleeding due to these causes are : (1) The menstruation may occur every third or second week, or even weekly, though in point of quantity it may or may not be increased. The flow, moreover, may be very readily excited, as by a hot bath, or after a day of unusual exercise. (2) The general symptoms after a time point to anæmia, combined with the symptoms of the constitutional cause in operation.

§ 350. Hæmorrhage may, secondly, be due to a UTERINE FIBROID. The symptoms vary with the position of the tumour. These tumours may be submucous, interstitial, or subserous. When the fibroid is submucous or interstitial, the symptoms of uterine fibroid are (1) menorrhagia and metrorrhagia. (2) Leucorrhœa and sometimes dysmenorrhœa are present. (3) On examination with the sound the uterine cavity is found to be enlarged ; and (4) on bimanual examination enlargement of the uterus, which is usually hard and bossed from the presence of more than one fibroid, can be detected. There is a tendency for the submucous variety to become polypoid, remaining attached to the uterus by a pedicle. The *subserous* fibroid may present no symptoms at all for many years and may even then be discovered by accident. Amenorrhœa may accompany such cases quite as often as menorrhagia, and the latter is never profuse. In short, pressure symptoms may be the earliest indication of a subserous fibroid. In uterine fibroids of all kinds the rate of growth, though it varies somewhat, is nearly always slow ; but as the tumour increases we get symptoms of pressure upon the surrounding organs, such as frequent micturition, varicose veins, neuralgia in legs and back, indigestion, difficult respiration, or hydronephrosis. Fibroids, especially when of large size, have a tendency to undergo degenerative changes which give rise to symptoms of toxæmia.

UTERINE POLYPUS is another cause of irregular hæmorrhage. They are of three kinds. The most common forms are fibroid polypi and mucous polypi. Placental and fibrinous polypi occur, the first after labour or abortion, arising from retained portions of the placenta, the second from the stump of a growth previously removed.

When very small, polypi can be made out with certainty only by dilating the os and exploring the interior. Later on, examination with the speculum may reveal the polypus hanging from the os into the vagina. After a time it may slough, and cause an offensive discharge.

§ 351. SUBINVOLUTION, or the non-return of the uterus to its normal size, is a very frequent case of menorrhagia after labour or abortion. After a confinement the uterus begins to diminish in size, and at the end of about two months resumes its normal length of 2½ inches. In cases of subinvolution we find (1) on vaginal examination that the uterus is enlarged ; (2) it tends in most cases to be retroverted and lower than normal ; (3) the patient generally complains of backache, bearing-down pain, and leucorrhœa ; and (4) lassitude, weakness, and general malaise are usually present.

The *Causes* of subinvolution are important : (1) Varying degrees of toxæmia occurring during pregnancy or the puerperium. (2) retained membranes or portions of placenta ; (3) pelvic inflammation ; (4) delayed labour or over-distension of the uterus ; and (5) the practice of not suckling the infant, account for this not infrequent condition, and therefore it is more often met with in those who have had numerous and rapid pregnancies.

§ 352. FIBROSIS, or CHRONIC METRITIS, is a condition in which the uterine tissue is thickened, tense, and hard or flabby. Profuse menorrhagia is the chief symptom ; there is usually a feeling of weight, and dysmenorrhœa, and the uterus is felt to be enlarged and firm. It is caused by infection, usually of gradual onset, and may occur at any age.

§ 353. THE MENOPAUSE, or climacteric, is the epoch at which the sexual activity of the female undergoes involution, when the menses, which are the sign of that activity, cease. This may take place in three ways : (*a*) They may cease gradually, and more or less irregularly ; (*b*) quite suddenly ; (*c*) they may be attended by a series of hæmorrhages. The last method, which is quite as frequent as either of the other two, is the one with which we are now concerned.

The existence of this cause of menorrhagia or metrorrhagia can only be recognised by the attendant phenomena. (1) The age of the patient varies considerably between thirty-five and fifty-five, the average being about forty-five. (2) The occurrence of "flush storms," which consist of a hot stage, a cold stage, with or without shivering, and sometimes a stage of perspiration. (3) Other nervous phenomena which may occur at this time are extremely varied. There is generally an irritability and restlessness, and generally also a marked tendency to depression of spirits causing the patient to burst into tears at the slightest provocation. This may amount to definite melancholia, especially when there is mental heredity. Sexual perversions, with a marked tendency to excess of all kinds, are apt to occur. (4) While fibroids and other gross lesions sometimes undergo involution at this epoch, carcinoma, if there be a predisposition, may make its appearance, and the case should be carefully watched from this point of view.

§ 354. MALIGNANT DISEASE of the uterus is clinically met with in four forms : (*a*) Cancer of the cervix, chiefly met with in multiparæ, between the ages of twenty-five and seventy ; (*b*) cancer of the body, which is chiefly met with in nulliparæ, between the ages of fifty and sixty ;

(c) sarcoma of the uterus, which is rare, unless we include under that term certain fibroids which appear to take on the malignant features of spindle-celled or large round-celled sarcoma; and (d) deciduoma malignum, a very rare form following parturition.

The symptoms differ in the first three varieties. (a) CANCER OF THE CERVIX usually runs a somewhat rapid course. (1) On digital examination the os has a hard, friable, granular feel, which is so characteristic that this feature and the blood-stained discharge upon the finger are alone, in experienced hands, sufficient to diagnose the disease. (2) In a later stage examination reveals a mushroom-like growth (" cauliflower ex-crescence ") hanging down into the vagina, readily breaking down and readily bleeding. It has a tendency to spread to the vaginal wall, to the utero-sacral ligaments, broad ligaments, and body of the uterus, leading to a fixity of the uterus and hardness which is easily made out on palpa-tion. (3) Metrorrhagia and menorrhagia are present. (4) In the intervals between the marked hæmorrhages there is a continuous watery leucorrhœa of pinkish-brown colour, often with a very offensive odour. (5) Local pain is usually a late symptom, but, like the wasting and the cachexia, is sure to supervene sooner or later.

(b) CANCER OF THE BODY of the uterus is chiefly met with in nulliparæ over fifty years of age. Bleeding occurs at a later stage than in cancer of the cervix. The symptoms are : (1) Metrorrhagia, and in the intervals pinkish brain-like matter is discharged ; (2) on bimanual examination the uterus is found to be enlarged. (3) If the passage of a sound is attempted, considerable hæmorrhage may take place. It should not be used in cases with much bleeding and offensive discharge. (4) Later· on, as the disease extends to the broad ligaments, the uterus becomes fixed ; this fixity to the educated finger is very characteristic of the disease. (5) The cachexia and other general symptoms resemble those of cancer elsewhere. The diagnosis from senile endometritis or a degenerating fibroid can be made only by microscopic examination of the discharge or a scraping taken for the purpose.

(c) SARCOMA OF THE UTERUS is a relatively rare condition. Its symptoms do not differ materially from those of uterine fibroid, except in the rapidity with which the case progresses, and the liability to deposits elsewhere.

§ 355. Extra-uterine Pregnancy (or Ectopic Gestation) may become manifest by menorrhagia, metrorrhagia, or amenorrhœa. The term is applied to the condition where pregnancy takes place outside the uterus, as a rule in the Fallopian tube, but sometimes in the ovary. The tube usually ruptures at the second or third month after fertilisation, either into the broad ligament (extra-peritoneally) or into the peritoneal cavity. The ovum in some instances is extruded through the abdominal opening of the tube into the peritoneal cavity, forming the so-called Ectopic Abortion.

Symptoms.—(1) In many cases paroxysmal pains are experienced in one iliac fossa ; (2) in about 70 per cent. of the cases there is a history of amenorrhœa for some weeks or a month over time, followed in most cases by a history of irregular hæmorrhages from the uterus. A membrane or cast may be discharged from the interior of the uterus at the same time. (3) Other symptoms of early pregnancy, such as morning sickness, are but rarely present. (4) On bimanual examination a swelling is found

in the fornix, and the cervix is soft as in early pregnancy. In most cases, however, none of the above symptoms may be noticed by the patient, and advice may not bo sought until the time of rupture of the tube, when the patient consults us for *severe pain* and *hæmorrhage.* Extra-peritoneal rupture is attended and followed by the symptoms of pelvic hæmatocele; intra-peritoneal rupture by the symptoms of perforative peritonitis (§ 193). If the rupture takes place about the fourth week the shock is not so severe, and the hæmatocele often remains extra-peritoneal. The prognosis and treatment are discussed under Hæmatocele (§ 361).

The *Prognosis of Hæmorrhage* depends upon the cause in operation. · Uterine bleeding of itself is not fatal to life, but some forms are very intractable, and lead to considerable anæmia, debility, discomfort, and inability to fulfil the duties of life. (1) The undue bleeding at the MENO-PAUSE and of SUBINVOLUTION tends to spontaneous recovery, and that which is due to CONSTITUTIONAL conditions is usually amenable to treatment; so also, in many cases, is that due to PELVIC INFLAMMATION, or such cases may develop CHRONIC METRITIS. (2) METRITIS is one of the most intractable causes, though it responds to local treatment. (3) The prognosis in a case of FIBROID tumour depends upon its position. The submucous varieties (and mucous polypi, § 350) are readily treated, but if neglected these may slough, and produce death by exhaustion and septic intoxication. The subserous form may give but little trouble for a great many years, and then chiefly by pressure symptoms. The interstitial form is the most serious, and if there be much loss of blood and consequent prostration the patient can only live a life of invalidism. When of large size these tumours are very difficult to treat. Fibroids, even if recurring near the menopause, should be treated surgically, because they rarely spontaneously disappear. If the bleeding is not yielding to styptics, removal of the tumour should be performed unless the patient's general condition is prohibitive. Curettage is not permissible. X-ray treatment has been given with much success in cases which refuse operation. (4) CANCER is the most serious of all the causes of hæmorrhage. Cancer of the body of the uterus is not so grave as cancer of the cervix. The chance of recovery depends upon the diagnosis of the disease and its treatment surgically *at an early stage.* If cancer of the cervix is discovered before it has spread to the parts around, or if cancer of the body is taken in hand while the uterus is still freely movable, operation offers a fair prospect of recovery. The prognosis of extra-uterine pregnancy is discussed in § 361.

Treatment of Hæmorrhage.—(a) Symptomatic, in all forms. To relieve the hæmorrhage calcium chloride in large doses (20 grains (1·2) or more) is most useful, as it promotes the coagulability of the blood. Ergot, adrenalin, dilute sulphuric acid, tinctura hydrastis, tinctura hamamelidis, styptol, tonics, chloride of iron, quinine, nux vomica, are all useful. 1 c.c. of a 20 per cent. solution of pituitary extract may be injected intramuscularly or adrenalin applied locally in severe cases. If the hæmorrhage is alarming and plugging the vagina has been tried, the uterus must be plugged after dilating the cervix. (b) Remedial treatment is directed to the cause, and must be adopted in addition to the foregoing. (c) In all cases general

measures are required—the food must be nourishing, exercise must be avoided near the period, and the patient must rest in bed while the flow is profuse. While strong purgatives on the one hand must be avoided, it is extremely important, on the other hand, to avoid constipation. For the menorrhagia of the menopause bromides and calcium chloride are recommended. Pituitary extract by the mouth, alone or with thyroid, according to the other symptoms present, aids all relaxed conditions and the menopause. X-rays in expert hands cure interstitial fibroids, and copper ionisation cures metritis in the young and at the menopause.

§ **356. Amenorrhœa** is that condition in which the catamenia are either deficient or absent. The term *primary* amenorrhœa is applied to the condition in which menstruation has never occurred, as in rare cases where there is a congenital absence of the organs concerned in the function, and also in cases of infantile uterus and undeveloped ovaries. *Apparent* amenorrhœa is that form in which there is a feeling of fulness in the breasts and abdomen every month, but the menstrual flow is retained behind an imperforate hymen, an occluded os or vagina. In *secondary* amenorrhœa, the flow, after having been once established, ceases or becomes deficient for a time. *Physiological* amenorrhœa is the cessation of the menses which occurs in pregnancy, a fact which must always be borne in mind even amongst the most irreproachable patients.

IN PREGNANCY, the physiological cause of amenorrhœa, the *General Symptoms* are as follows : (1) Morning sickness is usually one of the earliest, coming on about the first or second, and ceasing at the fourth month ; (2) the mammæ present a dark areola around the nipple, they become enlarged and after the third month contain colostrum. The *Local Signs* are : (1) On digital examination there is *a softness* of the os which is unmistakable to the educated finger ; (2) a gradual increase in the bulk of the uterus is early apparent. These are the earlier symptoms. From the third and fourth month we have a series of unmistakable signs—viz., (3) about the eighteenth week fœtal movements can be felt by the physician, and (4) the fœtal heart-sounds (at the rate of 120 to 150 a minute) can be heard on auscultation, usually midway between the umbilicus and either anterior superior spine ; and (5) ballottement can be made out about the fifth or sixth month.

The *Causes* of SECONDARY AMENORRHŒA may be divided into constitutional and local causes. (*a*) *Constitutional* causes are by far the most frequent, especially anæmia, or chlorosis, and phthisis. It also occurs after severe illness, mental shock, and during prolonged lactation. Derangement of the ductless glands, as in Graves' disease and myxœdema, may lead to amenorrhœa. (*b*) The most important of the *local* causes is perhaps an ovarian tumour, in which the state of the catamenia varies, but the flow is often absent or irregular. Other causes are a chill during menstruation, inflammatory conditions in the pelvis, superinvolution of the uterus, and extra-uterine fœtation.

Treatment in constitutional causes consists in plenty of fresh air, exercise,

good food, and general healthy living combined with iron tonics. Warm baths, especially warm hip-baths at the expected time, are useful. It is very important to keep the bowels regularly acting, and the old-fashioned remedy of the aloes and iron pill is most beneficial. Permanganate of potash in 2-grain (0·13) pills has been recommended. It is always advisable to adopt general tonic treatment in young unmarried girls, and it is only after these have failed that local causes should be investigated. Pituitary extract is very useful in some cases, apparently stimulating the ovaries. Hormotone and other pluriglandular preparations are also beneficial.

SUDDEN SUPPRESSION of the catamenia is a form of amenorrhœa which requires special treatment. The flow has probably come on normally, and then suddenly ceased on the second or third day, and the patient suffers a good deal of general discomfort. In such cases the patient should put her feet in hot water or a mustard bath, or sit in a warm hip-bath, and then should get into a thoroughly warm bed with hot bottles and take hot drinks. Subsequently saline purgatives in constant small doses, and general attention to the health are indicated. When the time of the expected period again comes round, the procedure just mentioned should be adopted.

§ 357. Pelvic Pain.—Pain in and about the pelvis is one of the commonest symptoms of disorder of the female reproductive organs. " Bearing down " is often spoken of ; and " backache " or pain over the sacrum is so constant a feature of uterine disorders that it has come to have that association in the minds of the laity. The position and character of pelvic pain vary with the different maladies, but its degree is largely influenced by the temperament of the patient. Reference has already been made to painful menstrual periods (dysmenorrhœa), but the causes of a continuous pain (without reference to the menstrual period), such as that now in question, may be conveniently grouped into (*a*) those pains which come on more or less suddenly (acute conditions), and (*b*) those which come on more or less insidiously (chronic conditions). It must, however, be remembered that no hard and fast rule can be laid down in this respect.

(*a*) *The* **pelvic pain** *came on* **acutely** *and recently ; it is accompanied by more or less* CONSTITUTIONAL DISTURBANCE—PERI- or PARA-METRITIS, INFLAMMATION of the UTERINE APPENDAGES, PELVIC HÆMATOCELE, ACUTE CYSTITIS, or some other INFLAMMATORY CONDITION within the pelvis, may be suspected, and the reader should first turn to § 358.

If the PAIN *has come on* VERY SUDDENLY *with* FAINTNESS *and* NAUSEA, turn first to PELVIC HÆMATOCELE, § 361 ; *if it be accompanied by* METRORRHAGIA, it is **suggestive** of MISCARRIAGE, or extra-UTERINE FŒTATION (§ 355).

§ 358. Perimetritis (Pelvic Peritonitis), which is one of the most frequent causes of pain, is an inflammatory condition affecting the peritoneal surfaces around the uterus and its appendages. Exudation may be present, and in chronic cases the adhesions lead to a matting together of the pelvic viscera. The *Symptoms* of ACUTE PERIMETRITIS are (1) acute severe pain across the lower part of the abdomen and sometimes also over

the sacrum ; (2) the abdomen is distended and tender to palpation, and a suprapubic mass of matted intestine may be felt. The patient lies on the back with legs drawn up ; (3) on examination, the vagina is found to be extremely tender ; (4) on vaginal examination forty-eight hours later a mass of exudation is found in the posterior fornix, pushing forward the uterus and rendering it less freely movable, though not so fixed as in parametritis ; there may be a blood-stained discharge. (5) The general symptoms consists of high fever and quick pulse, with vomiting.

In CHRONIC PERIMETRITIS (1) the pain is felt across the lower part of the abdomen, and is often greater on one side ; backache is usually present. The pain is constant, of a bearing down character, worse at the menstrual period ; (2) dysmenorrhœa and sometimes symptoms of endometritis accompany it ; (3) on examination the mobility of the uterus is found to be diminished, and thickenings, *chiefly in the posterior fornix*, can be felt behind the uterus, in which situation a kind of " roof " to the vagina exists. (4) The general symptoms consist of an inability to stand or to walk for any length of time ; and in severe cases chronic invalidism with mental depression or hysteria results.

Causes.—(1) Inflammation of the vagina or endometrium, especially that due to gonorrhœa, extending up by way of the Fallopian tubes is a common cause of pelvic peritonitis. After confinement or abortion acute pelvic peritonitis is often caused by extension of inflammation. (2) Appendicitis, torsion of an ovarian cyst, extension of pus from the surrounding tissues. (3) Chronic pelvic peritonitis may originate from infection by way of ovarian tumours, fibroids, cancer, or tubercle. *Prognosis* and *Treatment* below.

§ 359. **Parametritis (Pelvic Cellulitis),** another cause of pelvic pain, is an inflammation originating in the connective tissue of the pelvis adjacent to the uterus. This also may be acute or chronic.

The symptoms of ACUTE PARAMETRITIS are (1) pain across the lower part of the abdomen, usually *shooting down one leg ;* and the patient usually lies with *one leg drawn up to relieve the pain.* In some cases no pain is complained of at first. (2) On examination swelling and tenderness are made out in *one of the lateral fornices*, surrounding the cervix and fixing and displacing the uterus. No swelling is felt in Douglas' pouch (the posterior fornix) unless pelvic peritonitis is also present. (3) The general symptoms are those of fever, generally of a hectic type, with quick pulse ; in those cases where no pain is felt attention is drawn to the condition by the rise of the patient's temperature.

In CHRONIC PARAMETRITIS there are backache, dysmenorrhœa, frequently uterine displacement (due to the contraction of the inflammatory tissue), and symptoms of endometritis. When it results in suppuration the pus may make its way in various directions upwards or downwards.

Causes.—Parametritis usually follows labour or abortion in which injury to the cervix, vagina, or perineum has occurred, with consequent *entrance of septic matter.* Thus, injury by septic instruments may also

produce it, and clumsy attempts at procuring abortion form a not infrequent source. Apart from these, parametritis is practically unknown. Very rarely it may be due to infection from ulcerations, growths in the rectum or bladder, or appendicitis.

Course and Prognosis.—(a) In *acute perimetritis*, the acute symptoms should subside in a week ; if widespread adhesions are present, part of the exudation will be absorbed, and part will remain, giving rise to the symptoms of chronic perimetritis. *Chronic perimetritis* is often incurable. The prognosis will depend (i.) upon the extent of the inflammation, and (ii.) its cause. If it is the sequel to an acute attack with widespread adhesions the patient will probably have chronic pelvic pain, menorrhagia,
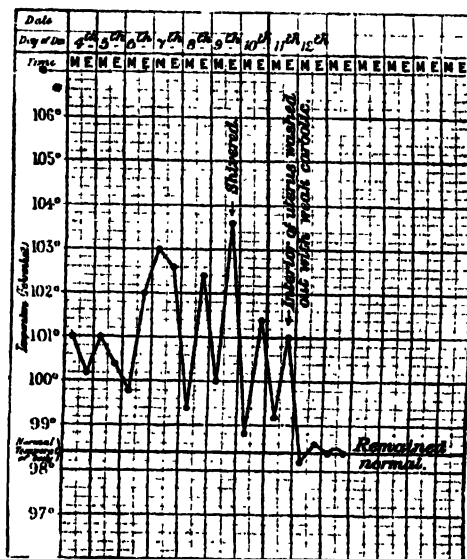
FIG. 88.—Chart of SEPTIC ABSORPTION showing effect of intra-uterine irrigation. Annie H., æt. 33, recently confined ; septicæmia seemed to be threatening, but after thoroughly washing out the interior of the uterus all the symptoms subsided.

leucorrhœa, and dysmenorrhœa all her life, with resulting chronic invalidism and nervous symptoms. Sterility is usual. If due to extension from a diseased organ, the patient will be subject to relapses with acute pain after any imprudence in the way of chills or over-exertion.

(b) In *acute parametritis*, if treated properly, the fever should subside in a week, and the exudation will probably be absorbed in three weeks. If the fever continues for four or five weeks pus has formed, and the patient will be invalided until the pus finds an exit (which may not be for months). The swelling felt in one lateral fornix becomes larger, pushing the uterus to one side, and later on a firm lump, which may extend to the iliac fossa, is felt along Poupart's ligament. The pus may point in the iliac fossa or follow the line of the vessels into Scarpa's

triangle; or it may burst into the vagina, bladder, rectum, or peritoneal cavity. In *chronic pelvic cellulitis* adhesions and fibrous tissue are formed rather than pus. They do not interfere with pregnancy. These may be absorbed in time, but anteflexion or version of the uterus is a common result of the contraction of the utero-sacral ligaments which occurs.

Treatment.—*Acute* peri- and para-metritis must be treated by (i.) absolute rest in bed; (ii.) hot fomentations, turpentine stupes to the abdomen, hot vaginal douches and vaginal ichthyol plugs; (iii.) saline purges; (iv.) morphia, if necessary, to alleviate the pain. In some cases (v.) vaccines or serum should be tried. Watch for the formation of abscess, and open it if possible by the vagina. *Preventive* treatment consists especially (1) in cleanliness of the hands of the nurse or doctor who attends a case of labour or abortion, and (2) in the curing of a vaginitis or an endometritis before it can extend up to the Fallopian tubes. The treatment of *chronic* peri- and para-metritis consists of (1) the administration of hot vaginal douches (up to 120° F.) daily, and of hypertonic saline, each douche lasting ten minutes; (2) ichthyol and glycerine tampons; (3) treating the pain, dysmenorrhœa, menorrhagia, and other symptoms as described under those conditions. Cold or damp and undue exertion in walking or standing must be avoided; and a certain daily interval of rest in the recumbent position should be ordered. If symptoms persist, surgical advice should be sought. During and after convalescence it is important to avoid constipation. When suppuration has occurred, the pus must be evacuated by free incision, preferably per vaginam. Vaccines may be very useful.

§ 360. Inflammation of the Uterine Appendages (viz., Ovaritis and Salpingitis) may also be a cause of pelvic pain.

OVARITIS is inflammation of the ovary, and should be distinguished from ovarian neuralgia. The *Symptoms* of ovaritis are so frequently accompanied by those of perimetritis that it is difficult to differentiate them. Indeed, *acute ovaritis* is found solely with acute peri- or para-metritis (*q.v.*). *Chronic ovaritis* may be recognised by (1) severe pain at the pelvic brim, extending down the thigh of the affected side; (2) pain increased by any pressure on the pelvic viscera (*e.g.*, by much standing, constipation, or flatus in the abdomen, and in severe cases by sitting); (3) menorrhagia and dysmenorrhœa, because endometritis so often accompanies ovaritis; and (4) dyspareunia. (5) The ovary is usually prolapsed, and therefore, per vaginam, a swelling, the size of a walnut, is found at the site of the ovary, to one side of or behind the uterus, acutely tender to touch, which causes a sickening pain. *General* symptoms, referable for the most part to the nervous system, very frequently supervene. The *Causes* of (1) acute ovaritis are sepsis after labour, abortion, or surgical operation; (2) chronic ovaritis may be due to the same causes as perimetritis, to alcoholism, to certain fevers (*e.g.*, mumps), or to infection after a chill, with suppression of menstruation.

SALPINGITIS (inflammation of the Fallopian tubes) occurs in three forms, hydro-, pyo-, and hæmato-salpinx. (i.) When the fimbriated end of the tube is closed by adhesions, the exudation within, unable to escape, tends to accumulate in the tube instead of escaping by the uterine opening (hydrosalpinx); (ii.) when the tubes are filled with pus (tuberculous, gonorrhœal, or septic) the condition is named pyosalpinx; (iii.) when the tubes are filled with blood, hæmatosalpinx.

The *Symptoms of salpingitis* are (1) pain across the lower part of the abdomen, usually greater on one side, often shooting down one leg ; (2) on examination a sausage-shaped swelling is found, usually double, running from the lateral fornices to Douglas' pouch ; (3) as perimetritis usually accompanies it, the uterus is less mobile than normal ; (4) dysmenorrhœa and menorrhagia are usually marked. (5) As regards the general symptoms—in hydrosalpinx there may be none, but pyosalpinx is accompanied by fever. In a pyosalpinx of sudden onset (gonorrhœal), the fever may be very high. *Causes.*—(1) Acute salpingitis is due to septic or to gonorrhœal infection extending upwards ; (2) chronic pyosalpinx, which is the commonest form of salpingitis in young single women, is usually due to tubercle, generally secondary to tubercle of the lungs or elsewhere. It may take an acute form. (3) A chronic or subacute vaginitis or endometritis extending upwards may result in salpingitis. Thus, sepsis or gonorrhœa following childbirth, and the use of dirty instruments, are common causes (see Vaginitis and Endometritis for other causes). (4) Hæmatosalpinx is due usually to a ruptured extra-uterine pregnancy.

The *Prognosis* of *ovaritis* depends on the extent of the inflammation around. If there is much matting the case is really one of perimetritis. If the inflammation is confined to the ovary the prognosis is favourable, provided the cause be removable and the patient is not of a neurotic constitution. In *salpingitis* sterility may result from adhesions closing the fimbriated extremity (though this cannot be diagnosed with certainty). Pyosalpinx is dangerous to life, as it may at any time burst into the peritoneum. Tuberculous salpingitis is very chronic, and less painful than the other forms. In all forms there is a tendency to relapse, and to peritonitis by extension rather than to spontaneous cure.

Treatment.—Acute and chronic *ovaritis* are treated like perimetritis (*q.v.*), together with hot applications to the hypogastrium when the pain is severe. Blisters and iodine applications over the iliac region have been recommended. Constitutional treatment must not be neglected—bromides, potassium iodide, and tincture of belladonna are beneficial. In *acute salpingitis*, when a pyosalpinx has resulted and the condition can be certainly diagnosed, some recommend laparotomy and removal of the tube ; others consider that rest in bed with hot douches will tide over the acute stage. In *chronic salpingitis*, rest, hot douches, and ichthyol and glycerine tampons may be tried for a period of two years at least. If this treatment and local diathermy fail, it will probably be necessary to remove the tubes.

§ 361. **Pelvic Hæmatocele** is an effusion of blood either into the peritoneal cavity (intraperitoneal) or into the connective tissue of the broad ligament (extraperitoneal), usually due to a ruptured tubal pregnancy (§ 355). Here there is a *sudden onset* of (1) severe pain, starting in one iliac fossa and soon spreading over all the lower part of the abdomen, accompanied by (2) faintness, perhaps unconsciousness, with (3) nausea, and in some cases vomiting. (4) There may be some uterine hæmorrhage, with discharge of a cast of the interior of the uterus. (5) On examination, the uterus, in the intraperitoneal variety of pelvic hæmatocele, is found pushed forwards behind the pubis, while in the extraperitoneal variety the swelling is smaller, and causes a lateral displacement of the uterus as in pelvic cellulitis. The intraperitoneal variety, if large, forms a lump which can be felt, on bimanual examination, both in Douglas' pouch and above the pubic, and the abdomen is tender and distended. After forty-eight hours, adhesions form and the uterus is fixed, and other signs of pelvic peritonitis may then ensue. The temperature begins to rise in twenty-four hours after the onset of pain—that is to say, when the pelvic peritonitis commences.

Diagnosis.—If the bleeding is (*a*) intraperitoneal, the hæmorrhage is rapid and excessive ; (*b*) if extraperitoneal, it is usually slow and limited in amount and tends to become encysted. (*a*) In the former, in addition to the symptoms of abdominal pain with collapse, there are the symptoms caused by hæmorrhage, viz., restlessness and air-hunger. The diagnosis from a *ruptured viscus* (§ 193) is very difficult at first. (*b*) When there is a smaller amount of bleeding, there may be acute pain and collapse, as above, but the symptoms may subside after a few hours, and attacks of pain may

recur at intervals for days. The local signs resemble *pelvic cellulitis*, from which it may be diagnosed by a history pointing to extra-uterine pregnancy, and by the fact that pyrexia is absent at the onset, and there is a pallor and a pulse of low tension.

Prognosis.—If hæmorrhage be large, death has been known to occur in about an hour. In smaller hæmorrhages adhesions due to pelvic peritonitis or cellulitis follow, and the exudation may be (i.) entirely absorbed, or (ii.) may go on to suppuration with a danger of general peritonitis. When due to extra-uterine pregnancy, an extra-peritoneal is not so immediately serious as an intraperitoneal hæmorrhage. Secondary rupture may occur into the peritoneum. In rare cases the fœtus may live till full time, when the patient goes through a spurious labour, after which the placenta becomes absorbed and the fœtus mummified, causing no symptoms.

Treatment is operative, except in the encysted variety, when operation is not so urgent.

(*b*) *The* **pain** *is of a* **chronic** *character, is of considerable duration, and is* UNATTENDED *by* PYREXIA. Almost any of the different diseases mentioned in this chapter may be suspected. Examination may reveal ENDO-METRITIS, ENDOCERVICITIS, CHRONIC PERI- or PARA-METRITIS, or a UTERINE DISPLACEMENT ; or careful bimanual examination may reveal a PROLAPSED OVARY or an INFLAMED TUBE. UTERINE DISPLACEMENTS and PELVIC TUMOURS alone remain to be considered. Prolapse of the uterus is a cause of dragging pain, especially in its early stages.

§ 362. Uterine Displacements.—The normal position of the uterus is one of anteversion, with slight anterior flexion. The uterus undergoes physiological displacements according to the fulness of the bladder and rectum. In itself a displacement leads to no symptom ; the symptoms so often associated with displacement are due in the majority of cases to the inflammatory processes in or near the uterus which have caused the displacement. Tumours and inflammatory exudation in the pelvis may cause LATERAL or UPWARD DISPLACEMENTS of the uterus.

FORWARD DISPLACEMENTS (ANTEFLEXION).—On bimanual examination the os is found to be high up, and the fundus is felt unduly far forward. In single women a stenosis of the os or an elongated cervix with spasmodic dysmenorrhœa may accompany a forward displacement of congenital origin. As above stated, *Symptoms* may be entirely absent, and attention is first drawn to the condition when other mischief, such as pelvic inflam-mation, endometritis, parametritis, or a history of dysmenorrhœa, sterility, or constantly recurring abortions, is present.

Causes.—(1) A congenitally ill-developed uterus is often displaced forwards. A forward displacement is diagnosed to be pathological in origin, as distinct from physiological, by the lessened mobility of the uterus, and the pain set up on attempting to move it. Forward dis-placements are found in association with (2) pelvic peritonitic adhesions, and (3) cellulitis affecting chiefly the utero-sacral ligaments.

Prognosis.—Anteflexion is a frequent concomitant of sterility. Its treatment is extremely troublesome, but if consistently and carefully carried out a radical cure is certainly to be expected unless the condition is due to a considerable degree of pelvic peritonitis or cellulitis, when the prognosis depends upon the removability of these conditions.

Treatment.—Treatment must be directed to any pelvic peritonitis or cellulitis present (*q.v.*). Ichthyol tampons and hot douches with purgative treatment will work wonders in the slighter forms. Massage is highly recommended where the anteflexion is due to the contraction of the utero-sacral ligaments. Dilatation of the cervix has aided some cases.

BACKWARD UTERINE DISPLACEMENTS consist of *retroversion* and *retroflexion.* In a backward displacement there is also a certain degree of descent of the uterus. Retro-displacements in themselves cause no symptoms; sometimes they are congenital. On examination the finger detects the forward displacement of the cervix, which is usually somewhat lower than normal. The uterus is not palpable in the anterior fornix, whereas a lump is felt in the posterior fornix, which is found to be the uterus because it is movable with the cervix, and can be felt to be continuous with the cervix.

Symptoms arise when pelvic adhesions are present, or when the displaced organ interferes with other organs in the vicinity. In such conditions, a retroverted uterus gives rise to (1) pain in the back and the lower part of the abdomen of a bearing down, dull, aching character ; (2) dysmenorrhœa and menorrhagia ; (3) constipation and painful defæcation. (4) If pregnancy occur, the sickness of the early months is excessive, and after the fourth month there may be retention of the urine, with dribbling, and subsequently sloughing cystitis.

Diagnosis.—The diagnosis of a backward displacement is not difficult, but the diagnosis of the cause may be obscure. It is important first of all to determine whether the uterus is freely movable or not, as the prognosis and treatment differ.

Causes.—The causes of backward displacement are (i.) congenital ; (ii.) the dragging of adhesions consequent on pelvic peritonitis ; (iii.) changes in the uterine tissues, such as subinvolution, or tumours in the walls ; (iv.) relaxation of the ligaments, as after pregnancy ; (v.) sudden fall or strain ; and in a few cases (vi.) a habitually over-distended bladder. Several of these causes may act in combination ; thus, subinvolution together with relaxation of the ligaments causes a retroversion with a certain amount of downward displacement of the uterus, as pointed out in Prolapse.

Prognosis.—(1) So long as the uterus is freely movable and not enlarged, there may be no symptoms until pregnancy occurs. Most often, perhaps, constantly recurring abortions take place. (2) In time retrodisplacements are apt to lead to congestion and enlargement of the uterine body, with endometritis, " erosions," and prolapse of the ovaries. Adhesions may ensue with chronic inflammation of the tubes and ovaries. (3) Where the uterus is bound down by adhesions, there is a condition which, according to Playfair, is " not fatal, but tends to life-long discomfort."

Treatment.—(1) Where the uterus is freely movable, replace it by bimanual manipulation. A Hodge pessary, if comfortable, should be worn so long as the uterus tends to return to the backward position.

Where there is pregnancy and the uterus cannot be replaced, even under chloroform, it may be necessary to terminate the pregnancy. If adhesions hold the fundus down, they must be divided. In the majority of cases in which pregnancy occurs in a retroverted uterus, spontaneous rectification of the fundus occurs between the third and fourth month. Pelvic inflammation must be treated, and tender prolapsed ovaries may require to be treated before replacement of the uterus.

§ 363. The following are some of the more important **Pelvic Tumours and Vaginal Swellings** : (a) *Internal tumours*—(1) uterine fibroid ; (2) cervical or uterine polypus ;· (3) cervical or uterine cancer ; (4) retroverted uterus ; (5) pelvic cellulitis ; (6) ovarian tumour ; (7) pyosalpinx ; (8) appendix abscess ; (9) pelvic hæmatocele ; (10) hydatid of the pelvis. (b) *External swellings* or swellings about the vulva may be due to (1) prolapse of the uterus ; (2) inversion of the uterus ; (3) prolapse of the vaginal walls (cystocele and rectocele) ; (4) cysts or tumours of the vaginal wall—e.g., of Bartholin's gland ; (5) uterine polypus with a long pedicle ; (6) local conditions of the *vulva*, such as abscess, hæmatoma, or labial thrombosis (§ 345) ; (7) *cysts of the vaginal wall* are usually found on the anterior wall, about the size of an egg and painless ; (8) hernia.

Most of these various conditions have already been fully referred to, but three conditions which may appear as external swellings remain to be described—PROLAPSE OF THE VAGINAL WALLS, PROLAPSE OF THE UTERUS, and INVERSION OF THE UTERUS.

§ 364. Prolapse of the Vaginal Walls is very common in multiparæ, especially of the anterior wall. It is then named cystocele, because of its close connection with the bladder ; indeed, the anterior vaginal wall may draw down the posterior wall of the bladder along with it. Prolapse of the posterior wall may occur, and when the rectum is prolapsed also, is named rectocele. But, as the rectum is not so intimately attached to the posterior vaginal wall, a prolapse of that wall is not usually a rectocele. The only symptom in addition to the swelling may be difficulty in passing water until the prolapsed part is pushed up. The diagnosis from a cyst of the vaginal wall is made by passing a sound per urethram and with one finger in the vagina, feeling the point of the instrument in the bladder. The chief predisposing cause of prolapse of the vaginal wall is a ruptured perineum.

For the *Treatment* of the two conditions, see below.

§ 365. Prolapse of the Uterus is its displacement downwards. Three degrees of displacement are described : (i.) The organ may occupy a position somewhat lower than normal ; (ii.) it may have partly or entirely passed through the vaginal orifice (procidentia) ; and (iii.) in extreme procidentia it lies entirely outside the vulva, the body lying in the inverted vaginal wall.

In slighter cases the vaginal wall is seen coming down on asking the patient to strain. In severer degrees the cervix can be seen and the body of the uterus and the ovaries can be felt. The other symptoms of prolapse of the uterus are : (i.) The uterus is enlarged, the cervix is frequently hypertrophied, there may be accompanying endometritis or endocervicitis ; (ii.) in early cases there may be incontinence or frequent micturition ; later, there is difficulty in passing water till the prolapsed organ is pushed up ; (iii.) sometimes there is a weight or a bearing-down feeling in the pelvis, but more often no pain is complained of, only the discomfort of the lump during walking and sitting. In the early stages, on the other hand, backache may be a prominent feature. (iv.) The uterus is usually retroflexed. (v.) Leucorrhœa is usually troublesome. Ulceration of the external parts is apt to supervene on procidentia.

Causes.—(1) The predisposing causes of prolapse of the uterus, as in prolapse of the vagina, are (i.) a ruptured perineum ; (ii.) a relaxed condition of the parts after labour ; and (iii.) a laborious occupation which demands much muscular strain, such as that of a washerwoman. The exciting causes are (i.) increased intra-abdominal

pressure, such as occurs with muscular work and tight-lacing; (ii.) the increased weight of the uterus in cases of subinvolution or tumour of the wall.

Treatment.—Preventive treatment is highly important. Every woman must rest sufficiently long after labour to ensure involution of the uterus. All perineal lacerations must be repaired as soon as possible. The uterus must be replaced by pushing up first the posterior vaginal wall, then the uterus, then the anterior vaginal wall. Then rest in bed, with tonics and general massage, may cure the condition. In other cases tampons of ichthyol and glycerine are inserted and changed every twelve hours; when inflammation or swelling has been reduced a ring pessary is introduced and should be cleaned every two months at least. In cases where procidentia has occurred a cup and stem pessary may be necessary. After the menopause prolapse may be difficult to cure, because a pessary in the vagina of old people is so apt to cause ulceration. Surgical interference may be required.

Inversion of the Uterus.—Sudden inversion of the uterus may occur in the third stage of labour, when the fundus is relaxed, but here we are concerned only with the chronic form of inversion, a very rare condition. It may be the sequel to acute inversion if the patient survive the shock, or it may be due to the dragging of a tumour. The fundus alone may be inverted through the os, or the whole uterus may be inverted. (1) The swelling is red, bleeds readily, and is tender. (2) The sound cannot be passed the normal distance, if at all. (3) Bimanually the fundus is found absent; and if a sound is placed in the bladder in the middle line and the finger in the rectum these can be made to meet without any uterus being felt. (4) There may be symptoms of bearing-down, menorrhagia, and leucorrhœa. The *Diagnosis* may have to be made from fibroid polypi; in which the fundus is not absent from its usual position. The orifices of the Fallopian tubes can sometimes be distinguished.

Prognosis.—There is no tendency to spontaneous cure. Death may occur after a long period of suffering and anæmia, from exhaustion or septicæmia. The *Treatment* is altogether operative, and we must refer the reader to a textbook on Gynæcology.

§ 366. It is proposed to discuss briefly the causes of the following symptoms for which the physician may be consulted : (*a*) DISORDERED MICTURITION (Retention, Unduly Frequent, Painful, or Difficult Micturition and Incontinence) ; (*b*) PAINFUL DEFÆCATION ; (*c*) PAIN ON SITTING ; and (*d*) DYSPAREUNIA.

(*a*) **Disordered Micturition** is dealt with more fully in kidney diseases (§§ 337 to 339) ; here only a few of those special to the female will be mentioned.

I. RETENTION OF THE URINE.—The *Causes* peculiar to women are impacted fibroids, malignant disease of the cervix involving the vagina, tumours of the vargina, a retroverted uterus (especially when about the fourth month of pregnancy), and other conditions causing obstruction of the urinary passage consequent on pressure over the mouth of the bladder. The condition is also found in reflex retention after operations on the perineum and in hysteria.

II. FREQUENT MICTURITION may be produced in women by (i.) pressure on the bladder from a tumour or an enlarged anteflexed uterus ; (ii.) a vascular caruncle of the urethra ; (iii.) acute cystitis ; (iv.) cystocele ; (v.) pelvic inflammation, especially during the early stages ; (vi.) calculi and gravel ; and (vii.) various nervous conditions.

III. PAINFUL MICTURITION is found especially in connection with urethral caruncle, cystitis, and in the early stages of pelvic inflammation or ovaritis.

IV. INCONTINENCE OF THE URINE is found (i.) in vesico vaginal or vesico-uterine fistula ; or (ii.) after dilatation of the urethra has been performed—*e.g.*, as a preliminary to lithotrity.

V. DIFFICULT MICTURITION is found (i.) after labour, when the parts are swollen and bruised ; (ii.) with prolapse of the uterus, in which case the symptom is relieved on pressing upwards the prolapsed parts ; (iii.) all causes of incomplete obstruction.

(*b*) **Painful Defæcation** may be due to (i.) retroverted and retroflexed uterus, especially when bound down by adhesions ; (ii.) an incarcerated retroverted pregnant

uterus ; (iii.) pelvic inflammation when acute ; (iv.) ovaritis ; (v.) prolapsed ovary ; (vi.) coccydynia ; and (vii.) a fibroid or other uterine tumours pressing upon the rectum.

(c) **Pain on Sitting and Coccydynia** are often associated with painful defæcation. (1) The commoner *external* causes of painful sitting are (i.) a vascular caruncle of the urethra ; (ii.) vulvitis and all other acute conditions of the vulva ; (iii.) hæmorrhoids or fissures of the anus. (2) The *internal* causes of painful sitting may depend upon (i.) an increased pressure within the pelvis—*e.g.*, pelvic inflammation, or any tumour within the pelvis ; (ii.) injury or inflammation affecting the sacro-sciatic and the sacro-coccygeal ligaments ; (iii.) a movable condition of the sacro-iliac joints after parturition ; or (iv.) a rheumatic condition of the same joints. (v.) Dislocation, inflammation, or " neuralgia " of the coccyx is also a recognised cause of the con-. dition.

Diagnosis.—The diagnosis of pelvic inflammation is treated of elsewhere. *Neuralgia* of the coccyx is known by the fact that the coccyx is sensitive to the touch. It may be connected with constipation or disorder of the rectum. Injury of the sacrosciatic or sacro-coccygeal ligaments is known by : (i.) the history of pain often dates from childbirth, or from the injury which produced it ; (ii.) pain is produced by pressure on the ligaments, which tightens them ; and (iii.) there is an absence of swelling or dislocation of the bone. *Dislocation of the coccyx* has no pain or tenderness, and is known by the fact that the bone, in most conditions, is displaced backwards. When the dislocation is found to be forward, it is much more painful, so that the patient usually sits on one ischial tuberosity—*i.e.*, sits sideways. In a *movable condition* of the joints there is a history of pregnancy with lameness towards the end of gestation, and the patient complains of pain over the pubic bone. In slight cases it might be very difficult to diagnose. *Rheumatism* is known by the absence of other local signs and by the shifting character of the pain, and perhaps the fact that the patient has other manifestations of rheumatism.

Prognosis and Treatment.—Vulvitis and pelvic inflammation are treated of elsewhere. Inflammation and neuralgia of the coccyx are usually cured by laxatives, hot baths, and sedative applications. Injury which has affected the ligaments may also be cured by laxatives and hot baths, but the improvement is slower. Some advise in extreme conditions the division of the ligaments. Dislocation of the coccyx, if backward, may be a cause of no great inconvenience, but if recent may be reduced at the time ; if of old standing it should be left alone. A forward dislocation, on the other hand, is much more troublesome, and may require the removal of the coccyx. A movable condition of the joints tends to recover spontaneously. It may be necessary to make the patient rest for a time, and afterwards to walk with a tight bandage across the pelvis.

(d) **Dyspareunia** (painful coitus) may arise from a variety of causes. (1) The most frequent is a functional spasm of the sphincter vaginæ, associated perhaps with a general neurotic state. In these circumstances the attempt to pass a speculum will sometimes elicit the same spasm, but may also be a means of cure. (2) Various other local conditions should be carefully looked for, such as a vascular caruncle of the urethra, vulvitis, or vaginitis (see above). Fissures or small ulcers between the folds of the parts, or hidden by the remnants of the hymen, are apt to be perennial causes of discomfort, which will remain undiscovered from month to month and perhaps year to year. (3) Ovaritis or a prolapsed ovary may produce considerable pain on deep penetration. (4) Parametritis (especially when associated with endo-cervicitis), perimetritis, and retention of foreign bodies, are also apt to become causes of dyspareunia. (5) Masturbation in the female. (6) Finally there may be, though this is relatively rare, a disproportion between the individuals concerned.

Prognosis and Treatment.—The condition of dyspareunia is apt to lead to considerable discomfort, not only to the individual, but to home life in general, and may lead to far-reaching consequences ; and when at length the aid of the physician is sought it behoves him to make his investigation with the greatest care, and express his opinion with considerable tact. The first step is to make a very careful and

minute examination in a thoroughly good light and under the most favourable circumstances for a local investigation, in view of the minute causes which may underlie the difficulty. The passage of a good-sized speculum will often cure vaginismus. The local conditions referred to must be treated. Cocaine ointment and suppositories and small doses of bromide may be tried. Childbirth frequently cures vaginismus and many of the causes mentioned.

§ 367. **Backache.**—Pain in the back may accompany various chest diseases; for these see § 83. We are here concerned with the pain in the lumbar region which is so frequently complained of, especially by ·women. The symptom is dealt with in the chapter on diseases of women, not because pelvic disease is always associated with backache, but because pelvic troubles are perhaps the most common cause of the backache for which the physician is consulted.

PHYSICAL EXAMINATION.—When the patient complains of backache, the physician should make a thorough examination of the region over which the·pain is felt. For the adequate performance of this examination it is essential that the patient should be stripped. If the clothes are removed only so far as the waist, important physical phenomena may be overlooked. Note first whether there is any curvature of the spine, displacement, tumour, or redness. By palpation endeavour to make out the presence and position of any tenderness or swelling. Examine next the precise position of the pain; whether it is unilateral or bilateral; whether it is accompanied by tenderness or not; whether it is aggravated by the movements of certain muscles or joints; whether it radiates along the course of any nerve. The presence or absence of muscular spasm should be ascertained. Examine the sacro-iliac joint and the costovertebral joints, and whether pressure over those joints elicits pain. An examination should be made next of the viscera; thus, percussion may reveal an abnormal area of dulness over the kidney; vaginal and rectal examinations may reveal disorders in these regions. The urine must be examined; it may show signs of kidney disease. Failing light from these sources, an X-ray examination should be made. The history of the onset of the pain, and of the concomitant symptoms at the time of the onset may give important clues in the diagnosis.

CAUSES OF BACKACHE.—(1) Backache occurs in many acute diseases, in most of the acute specific fevers, notably small-pox and influenza, and its cause is then recognised by pyrexia and other general symptoms.

(2) *Functional Causes.*—In nervous individuals, whose general health is below par, fatigue is usually evidenced by backache. It is frequently met with after childbirth, after infectious diseases, after operations, and associated with intestinal toxic conditions. In many of the last named there is marked tympanitic distension of the colon, and especially of the caecum. This is by far the most frequent cause of backache, both in men and women. It is relieved by rest, by suitable corsets, or other supports, and tends to disappear as the general health improves.

(3) Lumbago is known by: (i.) a history of a sudden onset, usually when stopping; (ii.) the pain is increased by movement of the lumbar

muscles, and is relieved by local warmth ; (iii.) tender points may be elicited in the fascia, near the origin and insertion of the muscles affected.

(4) Curvature of the spine, whether it be due to Pott's disease or to simple lateral curvature, is a cause of backache. The later stages of Pott's disease (tuberculosis of the vertebræ) show an angular curvature and come under the notice chiefly of the surgeon. The early stages are frequently overlooked, as no symptom may be present except pain. It demands for its cure prolonged rest and general treatment as in other forms of tuberculosis. The slighter forms of lateral curvature are a fre-. quent cause of backache in children and young women, especially on standing. This cause of pain often fails to be diagnosed, especially in the early stages, because of the neglect of the guardian or physician to examine the spine with the patient stripped.

(5) Sacro-iliac disease is another common cause of backache. It is known by : (i.) pain and tenderness over the joint is made out on palpation, or when the ilium is pressed inwards by the physician ; (ii.) pain is elicited by flexing the thigh on the abdomen while the leg is kept straight ; (iii.) the patient sometimes stands on one leg, and may complain of pain passing down one sciatic nerve ; (iv.) there is usually a history of strain. Strapping and fixation of the joint, with rest, relieve this form of backache.

(6) Osteo-arthritis is known by : (i.) signs of the disease elsewhere ; (ii.) the pain is made worse by coughing or sneezing ; (iii.) the pain usually radiates down the lumbar or sciatic nerve.

(7) Backache may be due to disease connected with the kidneys, such as perinephric abscess, pyolitis and pyelonephrosis, movable kidney, tumour and stone. An examination of the urine may first lead the physician to suspect the kidneys.

(8) Other abdominal tumours, such as retroperitoneal sarcoma, aneurysm, and tumour of the spine, cancer of the stomach and rectal disease.

(9) Gall-stones may rarely give rise to pain in the back before the pain works round to its usual position in front.

(10) Finally spondylitis or inflammation of the vertebral joints may be mentioned as a cause of backache sometimes following typhoid fever or syphilis. The condition is widely recognised in America, although it has received little attention in this country. The " *typhoid* spine " appears a variable time after typhoid fever. There is pain and tenderness, sometimes starting pains along the nerves, occasionally paresis and wasting. The diagnosis from peripheral neuritis and tuberculous disease of the vertebræ is made by finding the Widal reaction and albumin in the cerebro-spinal fluid. X-rays show osteo-periostitis. Kyphosis may result if the condition is not treated by rest.

CHAPTER XV

PYREXIA

MICROBIC DISEASES

WHEN a patient is suffering from some general or constitutional derangement, he complains of a vague "feeling of illness" (*i.e.*, malaise), or of "weakness" (debility, asthenia). He feels "generally" ill, and perhaps looks ill, but may be unable to mention any localising symptom, such as pain in the side or palpitation. Now, the first thing to do in such circumstances is to ascertain whether he is feverish or not, because all such conditions may be divided into two large clinical groups : A. **Debility with pyrexia,** which includes the Acute Specific Fevers and disorders in which there exists some localised inflammation ; and B. **Debility without pyrexia,** which includes the different forms of Anæmia and various toxic and nutritional disorders. The latter will be dealt with in Chapter XVI. In this chapter we are concerned solely with the various conditions attended by elevation of the body temperature.

§ **368. Definitions.**—The term **Acute Specific Fever** (or Specific Febrile Disease) has been applied to those fevers which are due to a specific or special poison, introduced into the body from without, and which run a definite course. If the poison was contracted from a previous case, but without contact with the patient, it was said to be an *Infectious* disease (*e.g.*, scarlatina) ; if the disease was produced only by actual contact with a person suffering from the malady, it was called *Contagious* (*e.g.*, syphilis) ; but these terms have always been used somewhat loosely and indifferently, and it would be better not to attempt any such distinction but to speak of them collectively as *Infective*. It would be out of place to enter here into the question of the nature of this poison ; but suffice it to say that there is direct or inferential proof in all the acute specific fevers that it is of microbic or parasitic origin. At first the microbes themselves were supposed to be the active agents of these diseases, but now in most cases the *causa vera* of the pyrexia and other symptoms is known to be a toxin or toxins which are produced by the microbe. This branch of knowledge has received enormous additions to it during the last quarter of a century (*cf.* §§ 416, *et seq.*).

The subject of Bacteriology will be referred to in a later chapter, and it will be sufficient to mention here the chief **clinical characteristics** which cause us to suspect a disease of being **microbic in origin.** They are four in number :

1. The occurrence of the disease in question in an *epidemic* form—*i.e.*, in the form of an outbreak, or as a series of cases which suggest that the patients contracted the disease either from one another or from a common source, the infection being conveyed to them through the air, the water, or other ingesta, or by the bite of an insect. Dietetic poisons (organic and inorganic) must be excluded.

2. Two features are common to all microbic diseases : (1) *Pyrexia* is present at some time during the course ; and (ii.) all the cases of disease run a more or less *definite course*—definite onset, gradual increase to an acme or fastigium, defervescence, gradual or sudden, followed by complete restoration to health, or death.

3. The constant presence in the blood, tissues or excretions of the patient of a *microbe* or *protozoon*, which is not there normally.

The *pathological proof* that a particular microbe is causally related to the disease consists in applying certain experimental tests (see § 416).

4. The fact that the attack is more or less protective against subsequent infection.

Epidemic, Endemic, and Sporadic are terms by which it is usual to express the relative prevalence of infectious diseases. A disease is said to be *Epidemic* when a large number of cases arise by infection from a common source or from one another at one time, followed by an interval in which none arise. Thus epidemics of measles, scarlatina, and diphtheria arise in the Metropolis and elsewhere from time to time. A disease is said to be *Sporadic* when it occurs only in isolated cases. Thus we speak of a sporadic case of mumps when no other cases of it have been known to occur about the same time and in the same district. An *Endemic* disease is one which is constantly present in a certain district. Thus enteric fever is endemic in London, ague in Central Africa and other marshy areas, and cholera in India.

PART A. SYMPTOMATOLOGY.

§ 369. Pyrexia and Symptoms which may attend it.—Pyrexia may in some instances be unattended by any symptoms, but in nearly all cases the patient whose temperature is elevated complains of feeling " chilly," or he may have shivering or rigors ; or perhaps he feels " burning hot." Headache, restlessness, and vague pains in the limbs and back are also common symptoms, in addition to the malaise or weakness. His skin is hot and dry to the touch, his pulse and respiration are rapid, his appetite is bad, tongue furred, and bowels confined, his urine scanty and high coloured. In severe cases of fever there is great prostration, considerable mental dulness, and there may be delirium, or the " typhoid " state. By these symptoms we suspect the presence of pyrexia, and the suspicion is confirmed, and the degree of fever ascertained, by the clinical thermometer (see below). The various STAGES through which microbic disorders pass and the three important symptoms or conditions which are apt to be met with in patients suffering from pyrexia—namely, RIGORS, DELIRIUM, and the " TYPHOID STATE "—will now be separately described.

§ 370. Incubation and other Stages of Acute Specific Fevers.—There is nothing more characteristic of microbic or specific diseases than the *definite course* which they run. It is a curious fact that a person does not develop the disease directly after he has been exposed to infection. The interval is called the stage of *incubation*. The patient may be quite well during this stage, or feel a little malaise. Its duration is variable in most diseases, and in each disease differs from another (table, p. 497). This period corresponds to the time during which a healthy person who has been exposed to infection needs to be isolated (placed in quarantine, as it is called), to see if he will develop the disease. A glance at the first column in the table will show that a period of THREE WEEKS will cover the incubation of all the eruptive fevers. The actual *invasion* or development of the symptoms of the disease is usually more or less abrupt, except in enteric fever, whooping-cough, and sometimes measles. An *eruption* appears upon the skin within the next four days (except in enteric fever)

in those diseases which develop a rash, and which are called on that account the Exanthemata.   The fever and other symptoms go on increasing until the *acme* is reached.   Finally the last stage—the stage of *defervescence*—supervenes, and gradually the patient convalesces.

TABLE XXI.—SHOWING INCUBATION, DATE OF ERUPTION, AND DURA-TION OF INFECTION OF THE PRINCIPAL INFECTIVE DISORDERS.

DISEASE.	INCUBATION PERIOD.	DAY OF DISEASE ON WHICH RASH APPEARS.	INFECTIOUS PERIOD, or period during which the *patient* need be isolated.
Varicella.	10 to 19 days, average 14.	The rash is usually the 1st symptom noticed.	Till all scabs have separated, 2 to 4 weeks.
Scarlet Fever.	1 to 5 days, average 2½.	2nd.	From commencement of illness till an indeterminate date, which varies in different cases. Average 5 to 6 weeks. Rhinorrhœa, and possibly otorrhœa, may retain infection for 6 months or more.
Small-pox.	12 days.	3rd.	From commencement till not a trace left of scabs or desquamation. Most virulent during vesiculation, pustulation, and scabbing. 3 to 8 weeks.
Measles.	7 to 14 days, average 10.	4th.	*Great* in early period *before* rash out. Till scaling and cough cease. Usually 2 *weeks*.
Rubella.	7 to 21 days, average 14.	1st to 4th.	7 to 10 days from commencement.
Typhus.	Rarely less than 12.	4th or 5th.	Probably 3 to 4 weeks.
Enteric.	3 to 21 days, average 10 to 14.	Average 2nd week.	Several weeks after pyrexia has ceased. "Carriers" may retain their infection for many years.
Dengue.	2 to 6 days.	Initial rash 1st day. Terminal rash 4th.	
Diphtheria.	2 to 6 days, or more.	None.	At *least* 21 days after disappearance of membrane and all throat mischief. Theoretically, so long as affected surface contains diphtheria bacilli.

The period of incubation of the other microbic disorders so far as we know is given approximately below.   This is important, as the duration of quarantine depends on the period of incubation.

Ague, 12 hours and upwards.
Anthrax, 2 or 3 days.
Gonorrhœa, 2 or 3 days.
Influenza, 3 or 4 days.
Plague, 3 to 7 days.
Glanders, 3 to 18 days.
Relapsing fever, 4 to 10 days.
Whooping-cough, 6 to 12 days.
Malta fever, about 9 days.
Erysipelas, 3 to 6 days.

Cholera, under 14 days.
Yellow fever, under 18 days.
Tetanus, under 24 days.
Mumps, 12 to 24 days.
Syphilis, 15 to 25 days.
Hydrophobia, 40 days or more.
Tubercle, probably some weeks.
Pneumonia ⎫
Septicæmia ⎪
Cerebro-spinal fever ⎬ unknown.
Infantile diarrhœa ⎪
Sprue ⎭

§ 871. **Rigors** often indicate the sudden onset of pyrexia. A rigor is an attack of shivering attended by elevation of temperature and great acceleration of pulse rate, rapidly followed (usually) by sweating and a fall in the temperature. Such an attack may vary widely in severity from a simple feeling of " chilliness down the back, like cold water," to a shaking of the whole body, so that the patient shakes the bed beneath him. Severe rigors occur typically and *regularly* in the course of malaria, also at frequent but *irregular* intervals throughout the course of septicæmia, and are occasionally met with in the course of typhoid fever. In childhood,. rigors are often replaced by convulsions.

1. First, ascertain that the shivering is not of purely nervous origin, because a trembling much resembling a rigor may occur as the result of pure fright or from slighter causes in nervous people.

2. Procure, if possible, a series of temperature records, because rigors occur in association with several conditions which can only be differentiated .in this way.

Causes.—The causes of rigors are very numerous, but they are best approached in a general way as follows :

(*a*) Coming on in a person *previously healthy*, one should always suspect the advent of some acute illness. In children the eruptive fevers are sometimes ushered in with either convulsions or rigors. In adults, pneumonia, peritonitis, pyæmia, the eruptive fevers, malaria or influenza may be suspected.

(*b*) *Septic Infection.*—When rigors *supervene in the course of an illness* of any kind, abscess or pent-up pus in some position should always be the first thing thought of. *Before the days of the thermometer the doctor used to rely upon shivering and sweating as an infallible indication of the formation of pus.* In a case of pleurisy with effusion, for instance, which has hitherto been serous, the occurrence of shivering indicates that the contents of the chest have become purulent (empyema). Similarly, a rigor occurring with otitis media suggests extension to the mastoid cells, or it may point to sinus thrombosis. Rigors occurring in a case of cardio-valvular disease indicate the occurrence of septic emboli, or the supervention of malignant endocarditis. Shiverings and sweatings are apt to occur during the course of tuberculosis and many other conditions mentioned under the Causes of Intermitting Pyrexia (§ 407). If no obvious cause for an attack of shivering appears, we may suspect some internal ulceration or suppuration, such as appendicitis, or ulceration in some part of the urinary, biliary, or alimentary canals. If the rigor is due to a collection of pus, there will be found a definite leucocytosis.

(*c*) Some *shock to the nervous system* may produce rigors. The passing of a catheter is often followed by a severe rigor, and sometimes the temperature goes suddenly up to 105° or 106° F., and as suddenly down again. Irritating substances in the alimentary canal may produce rigors reflexly. Sudden obstruction in the biliary or renal passages is often attended by rigors, followed by a feeling of heat and sweating, and the temperature may

go up to 105° F. (Murchison). Severe pain, as in hepatic colic, may be accompanied by rigor even when there is no fever. A rigor, too, may be set up by the intravenous injection of some antiseptic or therapeutic serum, such as diphtheria antitoxin.

(d) *Neurasthenic* and *hysterical* patients are very apt to have shivering attacks, but these are unattended by elevation of temperature. Attacks of shivering may also constitute a symptom of *vaso-motor disorder*. It is, for instance, a symptom of the reaction which follows, and often forms .part of the " flush-storms " chiefly met with at the climacteric -" flushes and shivers," as the patients call them. In these also there is no elevation of temperature.

The *Prognosis* and *Treatment* belong to the several causal conditions, but in any case the patient should be kept warm in bed with a hot-water bottle to his feet, and a full dose of opium, combined with bromide, to soothe the nervous system, and in septic or malarial cases 5 to 10 grains of quinine.

§ 372. **Delirium,** or incoherence of thought, is another symptom which frequently accompanies pyrexia. The older authors used to describe three varieties of delirium : (1) Delirium ferox, in which the patient is very violent and maniacal ; (2) typhoid delirium, in which the patient lies on his back muttering, with subsultus tendinum ; (3) delirium tremens, in which there is great sleeplessness, hallucinations and tremors, not necessarily due to alcohol. The nature of the delirium is not always constant in any given disease. For clinical purposes, the *causes of delirium* may be divided into two groups—FEBRILE and NON-FEBRILE. It is important, therefore, to take the temperature at once in every case of delirium. Alcoholic subjects and children, especially if neurotic, are predisposed to delirium when attacked with only slight fever.

a. Febrile Delirium, or delirium with elevation of temperature, may arise under four circumstances :

1. DISEASES OF THE BRAIN, such as tuberculous meningitis. This kind is generally accompanied by pain in the head, retraction, vomiting, intolerance of light, and paralysis of cranial nerves.

2. ACUTE LOCAL INFLAMMATIONS in other parts of the body, such as pneumonia. It is advisable, therefore, to examine all the organs of the body.

3. All the ACUTE SPECIFIC FEVERS are liable to be accompanied by delirium. The tendency, however, varies considerably, though it is usually directly related with the height of the temperature and the nervous stability of the individual. It is important to bear this in mind, because, as a prognostic indication, delirium occurring in a disease like measles or acute rheumatism, in which it is rare, has a much more serious meaning than when it occurs in pneumonia, for instance, where it is usual (see Table XXII). Occurring in acute rheumatism, it is generally an indication of pericarditis, endocarditis, or some other serious complication.

4. Certain cases of DELIRIUM TREMENS of a SEVERE KIND are accom-

panied by an elevation of temperature. Indeed, the prognosis in this affection may largely depend upon the temperature. We must be careful to exclude local inflammations in such cases, for they are apt to come on very insidiously. In the worst cases of ACUTE DELIRIOUS MANIA also the temperature may be considerably elevated (see *b* 6, below).

TABLE XXII.— SHOWING THE RELATIVE FREQUENCY OF DELIRIUM IN THE VARIOUS INFECTIVE FEVERS.

Frequent in—	*Occasional in—*	*Rare in—*
Confluent Small-pox	Remittent Fever	Influenza
Typhus	Yellow Fever	Mumps
Lobar Pneumonia	Small-pox (modified)	Dysentery
Enteric Fever (after 1st week)	Measles	Cholera
Meningitis	Relapsing Fever	Acute Rheumatism
Cerebro-Spinal Fever	Scarlet Fever	Malaria
Erysipelas		Diphtheria
Plague		Rubella
Malignant Endocarditis		Varicella
Septicæmia		

b. Non-febrile Delirium may arise under six conditions :

1. DELIRIUM TREMENS (Delirium e Potu) is, as just mentioned, usually unattended by elevation of temperature, and is undoubtedly the commonest cause of non-febrile delirium. It is recognised by the history, the muscular tremors, sleeplessness, and the characteristic hallucinations.

2. CHRONIC RENAL DISEASE, and especially chronic interstitial nephritis gives rise in its advanced stages to a muttering delirium or incoherence, which thus becomes a symptom of the gravest import, and generally heralds coma and death. The delirium is due to uræmia, and occurs in other renal diseases.

3. POST-FEBRILE DELIRIUM (Post-Febrile Mania).—During the convalescence of pneumonia, enteric fever, and other exhausting diseases, especially such as run a protracted course, and have been attended with a high degree of pyrexia, mental symptoms may develop. These symptoms, which—in most cases I have met with—make their appearance without any warning, give great uneasiness to the friends. Nevertheless, by means of good food, tonics, and fresh air, such mental symptoms will entirely disappear.[1] Before venturing on a prognosis, however, inquiry should always be made for any family history of mental disease, for a hereditary taint greatly lessens the chance of recovery. The condition is recognised by the history of the previous malady. Sometimes the mental derangement consists simply of loss of memory, especially for the names of persons and things, but more often the mind " wanders " and there are delusions.

[1] A recent case of this affection which I have seen was that of a lady, æt. thirty-nine, who, after a protracted illness with subacute rheumatism, developed mental symptoms which lasted for some three months, until the administration of opium gave her the necessary quiet, and she completely recovered. She had delusions, wanderings at night, and serious loss of memory. She always addressed me as " Dr. Devill."

4. Reflex Delirium.—Trousseau [1] mentions cases of children with intestinal worms who had delirium, and several cases are mentioned by the same author which were caused by the tickling of the soles of the feet. The transient delirium connected with the severe pain of childbirth is probably of the same nature. I am inclined to agree with Griesinger,[2] who says that " mental diseases caused by intestinal worms would be very interesting and more practically useful if they could bear a closer investigation." Nevertheless, the transient delirium or mania met with at the climacteric comes with some probability in this category, the reflex cause being situated in the generative organs.

5. Deliriant Drugs should always be suspected when delirium develops suddenly in a person in health, especially children in the country, in the absence of any of the foregoing causes. The most important are belladonna, hyoscyamus, cannabis indica, stramonium, and others of the solanaceæ,. antipyrin, camphor in rare cases, œnanthe crocata, cocculus indicus (with which beer used to be adulterated), poisonous fungi, and sometimes salicylic acid and its salts, especially if adulterated, when given in large doses. Morphia in some people invariably produces delirium.

6. Acute Mania sometimes comes on very suddenly, and, as previously mentioned, only differs from " delirium ferox " or maniacal delirium in not being referable to some bodily disease or toxic condition of the blood. We are enabled to identify this condition by (1) the temperature not as a rule being elevated; (2) by its affecting a person previously in good bodily health; and (3) the exclusion of any organic lesion by a careful examination, both of the nervous and other physiological systems. As regards the temperature there is an exception in the rare and serious condition known as " acute delirious mania," in which marked pyrexia is present.

Prognosis.—Febrile delirium is not necessarily a grave symptom when it is associated with a *disease in which its occurrence is usual*—*e.g.*, pneumonia—and especially when the cause is only temporary ; but its presence adds considerably to the gravity of a case if the occurrence of delirium is unusual (see Table XXII), for it indicates a very severe attack, or the occurrence of complications, or both. *Non-febrile* delirium is a grave symptom in chronic renal disease. The prognosis is serious as regards mental recovery in all patients who have a hereditary tendency to mental disorder. In acute mania the prognosis is very grave.

Treatment.—It is necessary to provide a nurse or attendant, and restraint may be called for. *Remedial Treatment.*—An ice-bag to the head for an intracranial inflammation ; good nourishing food for mania and post-febrile delirium ; a brisk purge for uræmia. Alcohol is indicated if the pulse is weak, but if it is strong and bounding, alcohol, as a rule, aggravates the condition. In every case of febrile delirium the effect of alcohol should be carefully watched, and its amount kept down as much ˚

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[1] Clinical Lectures : New Syd. Soc. Translation.
[2] Griesinger on Mental Diseases : New Syd. Soc. Translation, p. 197.

as possible. The *symptomatic treatment* consists of the administration of sedatives, such as chloralamid, trional, chloral, and the bromides. Of these the former acts best in most cases. Opium and morphia require caution. In delirium tremens, for example, it does a great deal of good in some cases by procuring sleep, but in others it only aggravates the maniacal condition. Periodical affusions of cold or ice-cold water often have a very steadying effect in this condition. In post-febrile delirium and other conditions where the brain is suffering from malnutrition, opium in small doses is a most valuable remedy, and may be given without fear if the kidneys are healthy.

§ 373. **The Typhoid State** may be described as a condition of unconsciousness (coma) or semi-consciousness attended by elevation of temperature and muttering delirium, due to a toxic condition of the blood. The name of this condition was derived from its frequent association with typhus, but it is met with in many other fevers. With reference to the question of pyrexia, it should be stated that the comatose condition, due to renal disease (uræmia), advanced liver disease, and various poisons (particularly opium), has sometimes been described as the typhoid state, but these are apyrexial conditions, and it is preferable to include only those with pyrexia. In short, the typhoid state corresponds clinically to a state of coma *plus* pyrexia and muttering delirium.

Symptoms.—The typhoid state is always secondary to some febrile condition, in the course of which it arises. The first symptom usually noticed is sleeplessness with delirium, generally of the muttering variety, but by and by stupor supervenes, which gradually deepens. The mental faculties are obscured, but the unconsciousness is not always so complete as one would imagine. The tongue is dry, brown, and rough, and sordes collect upon the teeth. The pulse is rapid, feeble, and irregular, and the heart-sounds distant. The respiration is usually rapid, but shallow. The pupils are dilated, but the patient does not see. Nevertheless, he looks about at imaginary objects—" coma vigil." Dysphagia may supervene, and is a very serious indication of profound stupor. Stertorous respiration only occurs in like circumstances, and is another grave indication. The profound disturbance of the nervous system is evidenced by prostration, restlessness, subsultus tendinum (muscular twitchings), floccitatio or carphology (picking at the bedclothes), unconscious evacuation of bladder and bowels, and, in extreme cases, convulsions. The temperature is elevated, its height and course depending chiefly upon the nature of the primary malady.

Diagnosis.—(1) The " *typhoid state,*" as above mentioned, may be distinguished from *coma* by the presence of pyrexia, and the absence of evidences of renal or liver disease, apoplexy, or other cause of the coma. (2) Certain acute *inflammations of the brain* are, however, attended by pyrexia, and offer considerable difficulty. This is particularly the case with tuberculous meningitis. The presence of optic neuritis, retraction of the head, paralysis of the cranial nerves on the one hand, and the signs

of the primary malady which has produced the typhoid condition on the other, are evidences upon which we can rely in many instances.

Causes.—Patients with an alcoholic history are predisposed to the development of the typhoid state. Renal fibrosis (chronic interstitial nephritis) offers a similar predisposition.

1. The ACUTE INFECTIOUS FEVERS are the commonest causes, and particularly typhoid and typhus fevers. The Typhoid State occurs as an ordinary symptom of a grave attack in the course of these two diseases and in some others (see Table XXIII). In another group of diseases it occurs only occasionally, and in others it is rare. If it arises in either of these latter groups, it indicates either (1) a very severe variety of the disease, or (2) some serious complications; and, in any case, that the patient is likely to die.

2. Certain other INFLAMMATORY or INFECTIVE DISORDERS with local manifestations may be attended by the typhoid state, such as acute lobar pneumonia, acute pulmonary tuberculosis, ulcerative endocarditis, acute meningitis, and encephalitis lethargica.

3. Certain acute IDIOPATHIC DISEASES may, in rare instances, be attended by the typhoid state, such as acute gout and very intense forms of delirium tremens. It is extremely rare in acute rheumatism, unless accompanied by peri- or endo-carditis.

TABLE XXIII.—RELATIVE FREQUENCY OF THE TYPHOID STATE IN
DIFFERENT DISEASES. ALCOHOLIC SUBJECTS AND PATIENTS WITH
GRANULAR KIDNEY ARE PREDISPOSED TO THE TYPHOID STATE.

Frequently met with, especially towards the end, in—	*Occasionally met with in—*	*Rare in—*
Typhoid (Enteric) Fever	Scarlatina	Cholera
Typhus	Measles with broncho-	Variola (modified)
Confluent Small-pox (unmodified)	pneumonia	Dysentery
Erysipelas (severe)	Cerebro-Spinal Fever	Malaria
Septicæmia (including Malignant En-	Anthrax (Internal)	Relapsing Fever
docarditis and Osteomyelitis)	Remittent Fever	Acute Rheumatism
Meningitis	Undulant (Malta) Fever	
Lobar Pneumonia		
Acute Miliary Tuberculosis		
Acute Glanders		
Acute Anthrax		
Remittent Fever		
Comatose and Hæmorrhagic Malaria		
Yellow Fever		
Plague		

Diagnosis of the Cause.—The clinical investigation should be conducted on the same lines as in cases of pyrexia. Is it due to *local* or *generalised* inflammation ? First, every organ in the body should be thoroughly examined so as to exclude local disorders. Secondly, we proceed to the diagnosis of the general fevers from one another, and, if possible, obtain a series of temperature records. In cases where the cause of the typhoid condition is obscure, septicæmia, especially with endocardial involve-

ment, should always be suspected, and its origin carefully sought.[1]

Prognosis.—The typhoid state, like delirium, has a less serious import in diseases such as enteric fever in which it is frequently met with. But it is always a grave condition, and indicates profound cerebral depression. Occurring in the course of scarlatina, erysipelas, or measles, it often indicates pulmonary or cardiac complication, and is proportionately serious. As regards symptoms, the profundity of the stupor is a measure of the intensity of the microbic toxæmia, and dysphagia, stertor, or convulsions are generally lethal signs.

The Treatment of a condition such as this arising in the course of so many diseases must necessarily vary, and our first duty is *to ascertain what disease is in operation.* It is, however, due in all cases to the effects of the toxin upon the central nervous system. The blood poison consists partly of the microbic toxins and partly of the excessive nitrogenous metabolism incidental to pyrexia. The indications are (1) to eliminate the poison by diuretics, diaphoretics, and aperients ; and (2) to stimulate and support the patient's strength by nutriment and stimulants. Alcohol was formerly given in large quantities. At the present day more reliance is placed on strychnine, which is best given by hypodermic injection. Dr. Murchison treated patients admitted on alternate days into the London Fever Hospital on opposite methods, and found that they recovered just as well without alcohol ; though, on the other hand, it did no harm. In practice, the state of the pulse and of the heart should be our guide. As regards symptomatic treatment, if the delirium be very violent, sedatives such as chloral or bromide are indicated if the heart will stand them. For this reason chloralamid is to be preferred. Opium should be avoided, as it prevents the elimination of the poison. For the treatment of Hyperpyrexia, see § 422.

PART B. PHYSICAL EXAMINATION

The clinical investigation of pyrexial disorders consists of (1) CLINICAL THERMOMETRY ; (2) AN EXAMINATION OF THE ORGANS ; and (3) BACTERIOLOGICAL INVESTIGATION.

§ 374. **Clinical Thermometry and Types of Pyrexia.**—The temperature is ascertained by means of the clinical thermometer.[2] The temperature of the body is usually taken in the axilla or the mouth. The temperature may also be taken in the rectum, where it may be $\frac{1}{4}°$ to 1° higher than

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[1] While I was Medical Superintendent at the Paddington Infirmary a young woman was brought in with all the symptoms of the typhoid state. The subsequent course of the temperature and the occurrence of sweating and rigors declared the disease to be septicæmia, which was traced to a pelvic origin. She died, and the case was brought home to a professional abortionist, who was sentenced to penal servitude.

[2] Owing to the shrinkage of the glass, all glass thermometers are apt after a time to read too high unless they have been stored for months or years before the scale is marked and zero fixed. A clinical thermometer, for instance, may, at the end of a year after manufacture, read a whole degree too high. Hicks, of Hatton Garden, has patented a process of annealing thermometers which obviates this error, and does away with the necessity of correction after prolonged storage.

in the mouth. The temperature in the mouth is usually higher than in the axilla, which is best regarded as the normal. In children the thermometer may be held in the groin-fold or " crutch," the thigh being flexed on the abdomen for the purpose. The normal temperature of the body varies between about 97·8° and 99° F.; average 98·4° F. It is highest about 8 P.M., and lowest about 4 A.M. It tends to be lower in old age and higher in infancy, especially after an attack of crying. The temperature is often subnormal after a loss of blood, during convalescence, in cardiac failure, and in all states of collapse. The latter is sometimes the direct result of toxaemia.

A temperature of 100° is regarded as slight fever.
     „   „   102°   „   moderate fever.
     „   „   104°   „   high fever.
     „   „   105° and upwards is regarded as hyperpyrexia.

THE TEMPERATURE CHART.—*Very little information can be derived from a single observation of a patient's temperature, and in all cases of pyrexia one must know the course which it runs from day to day and hour to hour.*
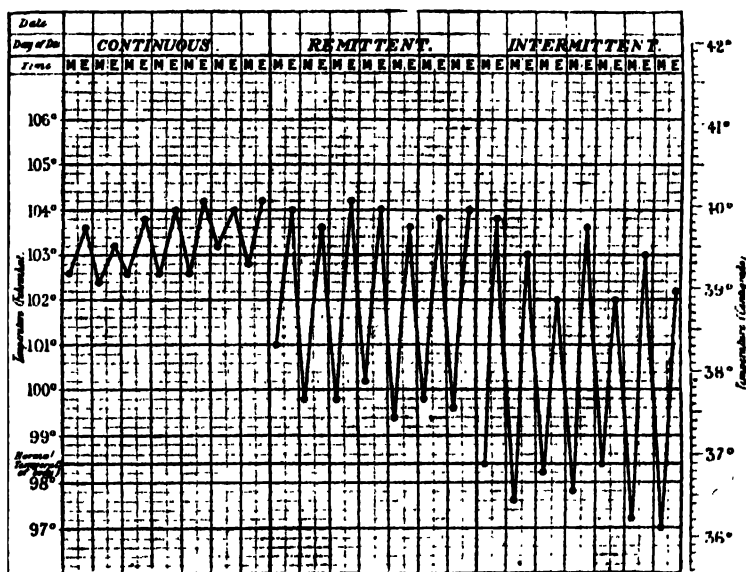
FIG. 89.—TYPES OF PYREXIA.—Continuous pyrexia showing only the normal variations in the morning and evening. Remittent pyrexia showing a drop of several degrees each day. Intermittent pyrexia where the temperature comes down to normal at some time every day.

In most cases of fever it is hardly possible to come to any conclusion without seeing a " chart " of the case—*i.e.*, a series of records. In all cases of pyrexia the temperature should be taken and recorded morning and evening; and in all acute cases it should be taken four-hourly. In cases of suspected tuberculosis and some other affections it is important to obtain hourly records throughout the day, otherwise slight elevations

may be missed. The pulse and respiration should also be observed, especially in abdominal inflammations, extensive broncho-pneumonia, and after severe attacks of diphtheria, where the temperature alone does not give us a true idea of the amount of mischief which is going on. In broncho-pneumonia the rapidity of respiration is often the most reliable indication. The onset of the pyrexia may be gradual, as in enteric fever or diphtheria, but more often it is sudden and may be accompanied by a rigor, as is sometimes seen in small-pox or pneumonia. Remember that the *onset is apt to be very sudden* in scarlatina, small-pox, and erysipelas ; it is *gradual* (taking perhaps two or three days) in measles and pertussis. During the next few days the temperature generally increases until the *acme* is reached. The termination may be gradual, when it is said to terminate by *lysis*, as in enteric ; or pyrexia may terminate suddenly by *crisis*, as in pneumonia and relapsing fever.

Types of Pyrexia.—In the absence of any eruption, the COURSE OF THE TEMPERATURE is our best, and may be our only, guide. It is usual to describe three types of pyrexia, according to the course which the temperature pursues from day to day (Fig. 89) ; (i.) *Continued or Continuous Fever*, where the temperature remains elevated for a considerable period, and where the *diurnal variation often does not exceed the normal diurnal variation*—viz., one, or at most one and a half degrees ; (ii.) *Remitting Pyrexia*, when the diurnal variation is greater than the normal diurnal variation, but where the temperature never comes down quite to normal ; (iii.) *Intermitting Pyrexia*, where the temperature at some time of the day is normal or subnormal, and at another time of the day, usually in the evening, it is raised one, two, or more degrees. But for clinical purposes the two latter may be grouped together, and thus we have TWO GROUPS of fevers—one in which the pyrexia is practically CONTINUOUS, and another in which there is a remission, or INTERMISSION, once or oftener during the twenty-four hours, usually in the morning.

The following are useful facts to remember regarding temperatures : (i.) The sudden advent of high fever in a previously healthy person without other symptoms indicates, in England, Scarlet Fever, Influenza, Small-pox, or Erysipelas, and sometimes Pneumonia. A very gradual advent is suspicious of Enteric Fever. (ii.) A fresh rise after the temperature has begun to fall indicates a complication or a relapse. (iii.) A sudden fall in the course of a fever (especially Enteric Fever) may indicate internal hæmorrhage, perforation of one of the viscera, or profuse diarrhœa. (iv.) A considerable rise in diseases usually non-febrile, such as tetanus, delirium tremens, cholera, cancer, epilepsy, apoplexy, etc., generally indicates a fatal termination.

§ 875. **Subnormal Temperature.**—The temperature of the surface of the body, as indicated in the axilla, is rarely more than one or two degrees below normal. When it is below 96° the condition usually amounts to collapse. Subnormal temperature is not so important, for purposes of diagnosis, as elevation of temperature ; but in the first four instances given below it may aid us in their differentiation. Subnormal temperature adds to the gravity of the prognosis in most wasting disorders. In regard

to treatment, temperature readings below the normal are indications for the administration of stimulants, nourishment, and the application of external warmth.

Causes.—1. Subnormal temperature as an indication of *lowered vitality* occurs in normal circumstances in the aged, in whom the temperature is habitually several fractions of a degree below normal.

2. A subnormal temperature is of considerable diagnostic significance in the *prodromal stage of tubercle*, and especially tuberculous meningitis. If a carefully recorded series of temperatures in a person suspected of tubercle show a subnormal morning and evening temperature (or *vice versa*), it adds to our suspicions.

3. The temperature takes a *sudden drop* in internal hæmorrhage or perforation of the bowels. In enteric fever this sudden fall may be the only indication of these serious complications. The rupture of an abdominal cyst, or of an internal organ, such as the spleen, liver, or kidney (very rare apart from injury), is attended by a sudden lowering of the temperature ; but these conditions are also attended by other and more distinctive signs.

4. In all severe *abdominal inflammations* prostration and collapse are marked features, and the temperature may in some cases be subnormal, although there may be considerable constitutional disturbance, as shown by the prostration, and the rapid pulse (§ 189).

5. Subnormal temperature occurs in several other disorders in which it is not of much diagnostic significance, because we depend upon other signs for their identification. Thus, the temperature of the body is lowered (i.) when there is an excessive withdrawal of heat from the body, as in cases of exposure combined with privation, or with extensive weeping skin eruptions ; or when large quantities of fluid are evacuated, as in severe diarrhœa or cholera (when the temperature may be 90° in axilla, though 105° in rectum) ; (ii.) in states of inanition or cachexia—*e.g.*, during convalescence from fevers, Addison's disease, cancer (especially of the alimentary canal), diabetes, and chronic mental disorders ; (iii.) when there is deficient oxygenation, as in cases of congenital heart disease, cardiac failure, alcoholism, jaundice, uræmia, pernicious anæmia, and acute yellow atrophy ; (iv.) in some diseases of the central nervous system, such as tuberculous meningitis, the onset of cerebral hæmorrhage, or cerebral tumour ; and (v.) in poisoning by phosphorus, atropine, morphia, carbolic acid, and other irritants.

6. In all states of **COLLAPSE** the temperature is considerably lowered (2° or more). Indeed, this is one of the chief means by which it may be distinguished from syncope.

§ 376. Examination of Organs.—All the viscera must be carefully examined in accordance with the Scheme of Case-taking, pp. 6 and 7, so that local causes for the pyrexia may be excluded. Examination of the urine or the stools may reveal an unsuspected cause of pyrexia. For *clinical* purposes there are two great groups of causes of pyrexia : (*a*) **local inflammations** such as pleurisy, appendicitis, abscess of the liver, etc., on the one hand ; and (*b*) **general bacteræmic** or **toxæmic conditions,** like scarlatina, rheumatic fever, and streptococcal or coli infection on the other.

If any local inflammation is found, turn to the chapter dealing with the disease of that part. But it must still be remembered that some constitutional disease (*e.g.*, some specific fever) *may* be present, of which the local disease is a complication. Thus pneumonia, which would be discovered in the course of our examination, is a frequent complication of enteric fever ; and endocarditis of rheumatic fever. There are two features which may lead us to suspect a combination of disorders such as this : (1) The signs and symptoms of the local disorder may be of an aberrant type (*e.g.*, see Aberrant Types of Pneumonia, § 102) ; and (2) the constitutional disturbance presented by the patient would be greater in degree or different in kind than would accompany the local disease if it were the only disease present.

§ 377. The Examination of the Blood often affords most valuable information, and it may be useful to make a blood-count or stain a film (§§ 427 and 428), to take a few drops of blood for the purpose of testing the Widal reaction, or to take a larger specimen of blood for bacteriological examination. For the Wassermann test about 5 c.c. are taken, usually from a vein in the forearm.

PART C. THE DIAGNOSIS, PROGNOSIS, AND TREATMENT OF MICROBIC DISORDERS

§ 378. Routine Procedure and Classification.—In cases of pyrexia we must investigate, as in other cases, three points :

First, THE LEADING SYMPTOM complained of by the patient will be one or more of those mentioned in § 369.

Secondly, THE HISTORY OF THE ILLNESS. The *date* when the symptoms commenced—*i.e.*, the PRECISE DURATION OF THE ILLNESS—is a most important matter. A few of the fevers —*e.g.*, enteric fever and diphtheria—commence insidiously ; but the majority are ushered in suddenly, very often with an attack of shivering (a rigor). Throughout the entire course of every case of fever the physician should have constantly in mind the " day of the disease," [1] so that he may know what events to expect at that particular period of the case. In enteric fever, for instance, on the fourteenth day, or a little later, the diurnal range of the temperature should commence to be more marked, and during the next few days special care should be exercised to avoid hæmorrhage or perforation.

Thirdly, THE EXAMINATION OF THE PATIENT comprises three important matters : (1) Physical examinations ; (2) is there, or has there been, an eruption ? and (3) the temperature and its course.

(1) EVERY ORGAN must be systematically examined (Scheme of Case-taking, pp. 6 and 7), and as carefully and thoroughly as the patient's condition will allow, in order that we may DETECT or EXCLUDE ANY LOCAL DISEASE. This is important, because all cases of pyrexia are associated with or due to some **local inflammatory disease,** or some **generalised febrile disorder** (*e.g.*, **enteric fever**), or both.

(2) WHETHER THERE IS OR HAS BEEN ANY ERUPTION is the next question. The first of the groups (*vide infra*) into which all fevers may be divided comprises those in which an eruption distinctive of the disease appears within the first four days (with one exception) after the illness. The day on which it appears in each disease should always be at our fingers' ends (table, § 497). •

(3) THE TEMPERATURE **and its course** is the next thing to investigate ; and it is of the greatest importance to obtain a CHART or succession of readings, after the manner described in § 374. The DURATION of the fever

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[1] Students do not always understand quite correctly the meaning of this phrase. For instance, the fourth day of a disease is the third day *after* its commencement. Thus the eruption of measles appears on the fourth day, and, supposing the patient were taken ill on a Monday, the eruption would appear on Thursday.

is of assistance in diagnosis, especially when it has lasted longer than two or three weeks.[1]

The **classification** of pyrexial disorders may conveniently be based upon the results of our examination—namely, the eruption, if present, and the course of the temperature.

GROUP I.—ACUTE EXANTHEMATA or ERUPTIVE FEVERS—*i.e.*, fevers which are characterised by AN ERUPTION distinctive of each disease appearing on one of the first four days of the illness (§ 379).

GROUP II.—CONTINUED FEVERS—*i.e.*, fevers in which the temperature runs a more or less continuous course, and which present NO ERUPTION during the first four days (§ 390).

GROUP III.—INTERMITTENT FEVERS— *i.e.*, fevers in which the temperature runs an intermittent (or remittent) course, and which present NO ERUPTION (§ 407).

If the physical examination reveals signs of disease of some particular organ, reference should be made to § 376, and to the chapter on diseases of that organ.

GROUP I.   THE ACUTE EXANTHEMATA OR ERUPTIVE FEVERS

In all the diseases in this group the onset of the pyrexia is more or less abrupt, and in the majority a well-marked GENERAL ERUPTION appears during the *first four days* of the illness. The course of the pyrexia varies considerably in the disorders in this group.

Common.		Rare.	
I. Chicken-pox (first day) ..	§ 379	VIII. Dengue (first day) ..	§ 386
II. Scarlet fever (second day)	§ 380	IX. Typhus (fourth or fifth day)	§ 387
III. Erysipelas (second day) ..	§ 381	X. Anthrax (first day) ..	§ 388
IV. Small-pox (third day) ..	§ 382	XI. Acute glanders	§ 389
V. Measles (fourth day) ..	§ 384		
VI. Rubella (first to fourth day)	§ 385		
VII. Enteric fever (usually eighth to tenth day), influenza, cerebrospinal meningitis, plague, and other members of Group II, occasionally present early rashes.			

In each of the acute exanthemata the ERUPTION has special and DISTINCTIVE CHARACTERS, which, together with the DAY OF THE DISEASE on which the eruption appears, may enable one to differentiate the members of this group from one another. SCARLET FEVER may be regarded as the type, but it will be convenient to take them in the order in which the eruption appears. TYPHUS is hardly ever seen, and DENGUE is not met with in England. ANTHRAX and GLANDERS are, like hydrophobia, derived from animals.

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[1] Excluding diphtheria and the exanthemata, it is found that the majority of short fevers, of a few days' duration, are due to " common colds," " rheumatism," " constipation," and " influenza." " Colds," including bronchitis, influenza, tonsillitis, and pharyngitis, 4,164 ; acute appendicitis, 1,504 ; acute arthritis, 1,016 ; salpingitis, 871 ; pneumonia, 803 ; lymphangitis, 365 ; sinusitis, 259 ; erysipelas, 241 ; poliomyelitis, 227.—R. C. Cabot, " Differential Diagnosis." London, 1911.

§ 379. I. **Varicella** (synonym: **Chicken-Pox**) may be defined as an acute contagious disease, manifested by an eruption of successive crops of limpid vesicles, usually accompanied by slight exacerbations of fever. It is in most cases a trivial disorder of childhood. It was differentiated from small-pox by Heberden in 1767, but its autonomy was disputed for nearly a hundred years later.

Symptoms.—The *rash is generally the first sign noticed*, though it may have been preceded by malaise, headache, backache and feverishness. A scarlatiniform or urticarial rash [1] often precedes or accompanies the characteristic eruption which consist of pink, slightly raised, ovoid, or somewhat pyramidal papules, which in the course of twelve or twenty-four hours become vesicular. The typical vesicle is at first a thin-walled, translucent, glistening bleb, containing a clear fluid, which after a day or two becomes opaque and cloudy. The vesicle, meanwhile, loses its tension and dries up into a scab, which finally separates within ten days or a fortnight, but rarely leaving any extensive scarring. Some of the papules do not proceed to vesiculation at all, the papular phase persisting. The essential feature of this eruption is that it comes out *in successive crops*, and consequently we see different stages of the rash on the same area of skin. This process rarely exceeds four days, and is often less. The rash starts on the chest and neck, and usually invades the whole body, including the face, but is sparse on the limbs, especially towards the distal extremities. It may invade the mucous membranes, the palate being most often affected. As in small-pox, the rash is most profuse in areas subject to irritation. The whole disease seldom lasts longer than ten days, and it may be so trivial as to pass unnoticed by the patient. The temperature rarely exceeds 103° F. A case ceases to be infectious after the scabs have separated. The period of incubation is fairly constant. Although the limits may be stated as from eleven to twenty-one days, it is usually about a fortnight.

Diagnosis.—Modified Variola is the chief disease from which it has to be differentiated, although this should not be difficult, because in small-pox (i.) the rash comes out definitely on the third day; (ii.) it does not appear in successive crops; (iii.) its favourite situations are the distal extremities; (iv.) the evolution of the pock is much less rapid; and (v.) the constitutional symptoms are very definite and characteristic. *Herpes* is distinguished by the limited area, and grouping of the vesicles. That there is some causative connection between herpes and varicella is unquestionable, though their relation to each other is at present not clearly understood. *Pemphigus* is distinguished by the size and chronic character of the blebs. *Dermatitis Herpetiformis* is distinguished by its chronic character, by the vesicles occurring in groups, and irritation is usually severe.

Etiology.—Varicella is essentially a disease of childhood, but adults are not exempt. It occurs in epidemics, for the most part, of limited

[1] J. D. Rolleston, " The Accidental Rashes of Varicella." Brit. Med. Jour. 1907, i., 1051.

extent, though it is endemic in London. One attack usually confers immunity, but there are many reported cases of second and even third attacks. Other infectious fevers predispose to it; attacks following scarlet fever are apt to be severe. The disease can be inoculated, though not so constantly as small-pox.

Prognosis.—An attack is usually over in a week or ten days, but it is apt, particularly in adults, to be followed by weakness, which indeed may be more troublesome than the disease itself. *Untoward symptoms,* such as gangrene and hæmorrhage into and between the vesicles, and from the mucous membranes are rare. Death may be due to a confluent attack. *Complications* are few in number, the chief one being impetigo.

·Treatment.— The itching is generally the chief trouble, and this may be relieved by chloral, baths, or calcium chloride. Sponging the surface with carbolic lotion, 1 in 20, is very useful. The child should be prevented from scratching the pocks, as it causes suppuration and consequent scarring. Quinine and arsenic are the best remedies for the resulting weakness.

§ **380.** II. **Scarlet Fever** (synonym : Scarlatina) used to be one of the most serious, and one of the commonest, of the eruptive fevers. It is still very prevalent, though its severity has undergone remarkable mitigation in this country during recent years. It may be defined as an infective febrile disease attended by inflammation of the tonsils, and a punctiform eruption on the skin, followed by desquamation. There are six characteristic *Symptoms.* (1) After a period of incubation which varies from one to five days, though usually two to three, there is a *sudden advent* of high fever. The occurrence of this sudden pyrexia is of itself extremely characteristic of scarlet fever, small-pox, and erysipelas, and, occurring in a child previously healthy, is always suggestive of scarlatina. Vomiting also occurs in 80 per cent. of the cases (Caiger).[1] The temperature gradually subsides to normal about the fifth or sixth day in mild cases. It does not, as in small-pox, subside when the rash comes out (Fig. 90). (2) A *sore throat* appears on the first day, with the fever, and gives a characteristic scarlet colour to the fauces. The swelling is greatest about the fourth day in simple cases. Sore throat occurs with several of the exanthemata. In scarlet fever it is the tonsils and pharynx that are affected (rarely the larynx); in measles the larynx is chiefly affected ; in small-pox both the larynx and pharynx are involved. The inflammation may become very severe, and is always attended with more or less glandular swelling. (3) The *eruption* is the next symptom, and it is generally remarkably regular in its appearance—twenty-four to thirty-six hours after the advent of pyrexia. It has two elements—a generalised red blush, disappearing on pressure, and a number of minute points slightly raised and redder than the surrounding skin. It appears first on the front of the chest, axillæ, and arms, and is last developed on the limbs,

[1] Dr. F. F. Caiger, in the article on this subject in Dr. W. H. Allchin's " Manual of Medicine." Macmillan, London, 1900.

affecting finally the hands and feet ; but on the palms and soles there is *no* punctate or macular eruption as there is in measles. The face is flushed, but has no punctiform rash either. It continues well marked until the fourth or fifth day of the disease, then declines, and is generally completely gone by the seventh or eighth day, except on the outer sides of arms and legs, where for several days there often remain a number of coarse injected papules. (4) The typical *strawberry tongue* is seen about the fourth day. It is due to the stripping of the fur, which leaves a bright red denuded surface, with marked fungiform papillæ. (5) *Desquamation* is apt to occur with any severe skin inflammation, but it is more charac-
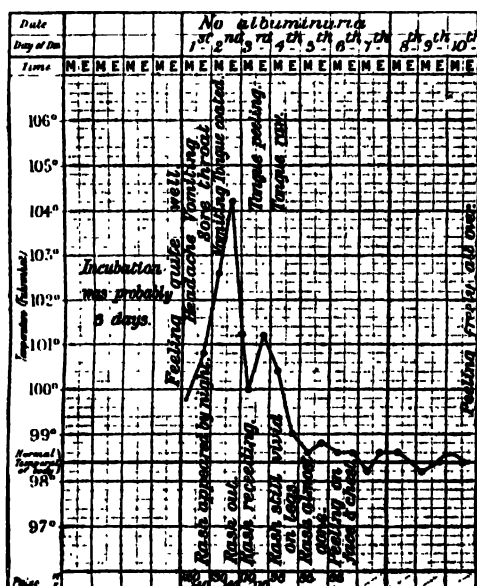
Fig. 90.—SCARLET FEVER.—Arthur M——, æt. 5. A typical mild case, specially as regards the initial symptoms, the rash, the tongue, and the desquamation. The various incidents are shown on the chart, for which the author is indebted to Dr. F. F. Caiger.

teristic in this than in any other fever. It begins about the fourth day, and continues for from four to six, or eight weeks—first on the face, and, following the order of the rash, *last on the palms and soles*, the complete desquamation of which may be very tedious. In the latter position the flakes are large ; elsewhere they are small and shreddy. (6) The *blood* shows a polymorphonuclear leucocytosis and moderate eosinophilia. According to some observers, the Wassermann reaction is positive during the acute stage, though this is denied by others.

Varieties.—There are, according to Dr. F. F. Caiger, three chief varieties : (1) The *Benign*, simple or ordinary type as above described. Various symptoms—*e.g.*, rash or sore throat—may be absent, and these cases are spoken of as *latent*. (2) In *Septic Scarlet Fever*, Scarlatina

Ulcerosa, or Anginosa, "the ordinary symptoms are aggravated by the presence of faucial ulceration, which, in addition to being a serious lesion in itself, provides a focus from which septic material is absorbed into the system" (Caiger, *loc. cit.*). Perforation of the palate may occur. The rash is often faint, but a blotchy or gyrate eruption frequently appears on the face and limbs in the second or third week. (3) In the *Toxic* form the patient is seized with high fever, delirium, and perhaps convulsions; the vomiting persists, the rash is very intense, but the throat symptoms, perhaps, ill-marked, and the patient dies during the first week. Toxic scarlet fever of such intensity as to deserve the name *Malignant*, in which there is low muttering delirium, usually a marked rash, and death without complications in a few days, is a very rare variety at the present day. In *Hæmorrhagic* cases petechiæ appear in the skin and mucous surfaces. These last are intense varieties of the toxic form.[1]

Diagnosis.- The diagnosis of scarlatina is not difficult in typical cases. The abrupt advent of high fever, accompanied by vomiting and sore throat in a child who has not had the disease, is always extremely suggestive, and if the disease is prevalent the diagnosis is almost certain. During the first few days the greatest difficulty is sometimes experienced in the diagnosis from *quinsy*, in which there is frequently albuminuria at the onset but less stupor and lethargy, generally less fever, and the history of previous attacks of quinsy. Slight albuminuria may be present in both conditions during the early stages, but in scarlet fever albuminuria is far more common towards the end of the third week, when it is the result of nephritis. Without the eruption it may be impossible to come to a definite conclusion, though the occurrence of vomiting in the early stage, the occurrence of rheumatism in the joints of the fingers or wrists towards the end of the first week, and the development of the "strawberry tongue" may assist the diagnosis. In doubtful cases it is best to act as if the graver disease were present (see Table X, § 130). *Diphtheria* has no punctate rash, though a flush may be seen on the chest and arms, but the characteristic membrane appears on the throat (see Table X) and the tongue remains coated. *Dengue* (*q.v.*) is accompanied by severe articular pains and a morbilliform eruption on the fourth day. The diagnosis is easier when the eruption is present. The scarlatinal rash is distinguished from the diffuse prodromal erythema of *small-pox* by the fact that the latter starts in the groins or axillæ, and that it invades the oral circle if the rash is diffuse, and lumbar pain is usually complained of. Enema rashes and *Epidemic Exfoliative Dermatitis* are sometimes mistaken for scarlatina. A *septic rash* may be scarlatiniform, but is distinguished by fever of a pyæmic type, and the presence of a septic focus, and by the absence of characteristic punctation. The erythema of *belladonna poisoning*

[1] *Surgical Scarlatina* is an unfortunate name suggested for an erythematous rash, accompanied by but slight constitutional symptoms, which sometimes occurs in surgical cases especially after tracheotomy, circumcision, wounds of the eye or burns. Such cases may or may not be scarlet fever.

is accompanied by great thirst, dryness of the fauces, and dilatation of the pupils. It is, moreover, unattended with pyrexia. *Copaiba rashes* and those due to so-called "*ptomaine poisoning*" may be a source of confusion.

Etiology.—Delicate children and puerperal cases have a strong predisposition to the disease. It is a highly infectious malady, especially at the outset and during early desquamation. The infection is propagated through the air for short distances, and carried by any article which may have become infected by the buccal or nasal mucus, such as a spatula, cup, spoon, fork, syringe-nozzle, or the nurse's fingers. It may attach to books and clothes, and is not infrequently conveyed by infected milk. The patient is generally regarded as infectious until desquamation has ceased, a period averaging four to six weeks, or even longer. There is no evidence, however, that the later desquamation of scarlet fever is ever infectious, traditional belief notwithstanding. On the other hand the infection is capable of surviving in the mucous discharges from the throat and nose for many weeks, and possibly the ears, long after the peeling is completely finished. One attack usually gives immunity for a lifetime, but by no means always. The disease is most prevalent during the autumn and early winter.

Prognosis.—It is always a serious disorder, because of the liability to complications, especially renal and ear disease. These dangers are avoided to some extent by keeping the patient in bed. Murchison used to teach that if a patient had been confined to bed three weeks, nephritis supervened less often. This point is of the greatest importance, as it is as likely to follow slight as severe cases. After the fourth week there is little danger of nephritis. The aggregate case-mortality under five years of age is about 5 per cent., but it varies in different epidemics. Over five it is less than 2 per cent., and is lowest between puberty and thirty years of age. The hæmorrhagic and malignant forms of the disease are those attended by great danger, although a septic attack in a young child is very likely to prove fatal. The danger varies with the malignancy of the symptoms, especially the throat symptoms, and the cardiac indications. Persistent vomiting indicates a severe attack. Delirium at night is more or less usual in bad cases, but violent delirium or stupor is a bad sign. A temperature of over 105° F. is a serious symptom. The disease often carries off the healthy and well nourished, and sometimes spares the delicate patient ; but in the *puerperal state* and in *tuberculous* patients the prognosis is very grave, since in the former case septicæmia is apt to supervene, and in the latter, an attack of scarlet fever is likely to reawaken a latent tubercle.

The *Complications* and *Sequelæ* are very important, for they may cause death, even after slight attacks. A considerable change has taken place in the nature of the complications and sequelæ met with in recent years, partly, no doubt, owing to improved methods of treatment. Acute ·nephritis and tonsillar or retropharyngeal abscess used to be regarded

as the chief dangers, but at the present day Dr. Caiger [1] gives otorrhœa and otitis media as the most important *complications*, attacking 15 per cent. of all cases, and leading, occasionally, to permanent defects in hearing, while mastoid abscess, septic thrombosis, suppurative meningitis and other septic conditions may ultimately follow if the ear disease be not cured. Next in order come simple albuminuria, attacking 7·9 per cent., definite acute nephritis, 4 per cent., together totalling 11·9 per cent., and cervical adenitis 11·4 per cent. Acute nephritis appears usually at the end of the third week, very rarely after the fourth, its advent being indicated usually. by hæmaturia, drowsiness, vomiting and dropsy. As a rule it clears up entirely. Both uræmia and the supervention of chronic nephritis are rare. Articular rheumatism 6·7 and secondary tonsillitis 3·1 per cent. are met with, chiefly among adults. The other proportions given by Dr. Caiger are ulcerative stomatitis, 1·7 per cent. ; and broncho-pneumonia, 1 per cent. Brawny swelling in the neck, cancrum oris, and noma pudendi are occasionally met with, and may need prompt surgical measures. Acute endocarditis and pericarditis rarely occur among the cases treated at the Metropolitan Asylums Board's Hospitals, whatever their incidence in cases treated in their own homes. Among the *sequelæ* subacute rheumatism, chorea and epilepsy are perhaps the chief.

Treatment.—The general treatment is dealt with in §§ 421 *et seq.*, but it will be well to make a few remarks on the symptomatic treatment. The throat is best treated by a chlorine gargle (F. 18). In children this may be applied by means of a syringe or a spray ; nitrate of silver and other caustic applications so long in vogue only aggravate the condition. For the glandular swellings apply glycerine and belladonna, or warm fomentations. Brawny swelling of the neck is a serious complication, and must be dealt with by early incision and frequent carbolic fomentations. For uræmic convulsions give a drastic purgative (croton oil) and a hot air or steam bath, supplemented, if necessary, by pilocarpine injections. Venesection may be tried if the convulsions persist, and the spasms may always be kept under with the aid of a few whiffs of chloroform. The patient should be kept in bed for three weeks, whether the attack be slight or severe, chiefly to prevent renal complications. The *hygienic* treatment is considered in §§ 419 *et seq.*, but a study of the long list of infective complications given above will show how important it is to treat this malady in a large and airy hospital instead of at home. The throat is in a highly vulnerable condition, and it is doubtless through this portal, or through the nose that the various infective *organisms* find entrance.

As regards immunisation, the recent work in the United States indicates that a hæmolytic streptococcus is the causal organism, on the following grounds : (1) inocu-

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[1] Dr. F. Foord Caiger. The complications of scarlet fever based upon an examination of 10,989 cases treated in the South Western Fever Hospital, Stockwell, during the years 1895–1904 inclusive.—Clifford Allbutt's *System of Medicine*, vol. ii., part i., p. 452.

lation of an apparently pure culture has produced scarlet fever in volunteers ; (2) intracutaneous injection of a filtrate of the culture gives a strongly positive reaction in susceptible subjects (Dick test) ; (3) preparation of a serum by immunisation of a horse with the scarlatinal type of *Streptococcus hæmolyticus* has a curative effect.

In toxic cases the subcutaneous injection of the serum of a convalescent patient (in 50 c.c. doses) has been tried. Others report favourably on the intramuscular injection of " whole blood " drawn from a scarlet fever convalescent, the amount drawn being 100 c.c. to 250 c.c. and immediately injected. This may not be easy to obtain, but is certainly worth trial, having regard to the hopelessness of the outlook in malignant attacks under ordinary methods of treatment.

§ 381. III. **Erysipelas** (synonyms : The " Rose," or " St. Anthony's Fire ") may be defined as an acute febrile contagious disease, characterised by a progressive marginated redness and tumefaction of the skin, usually attacking the face, or the neighbourhood of wounds. (1) *The Stage of Invasion.*—After an incubation period of three to six days the advent is abrupt, as in small-pox and scarlatina. The temperature on the evening of the same day may be 103° to 104° F., or more. Vomiting is very common, and so also are muscular pains, especially pain in the back,[1] like that of small-pox. (2) The *Eruption* begins about twenty-four to thirty-six hours after the advent of fever, as a red spot on the face or at the site of an abrasion (which may be microscopic). It often commences just within the external nares on one side at the junction of the skin and mucous membrane. It enlarges, spreads, becomes bright red, tender, and pits on pressure. The advancing edge is sharply defined and raised, the receding edge indefinite. The eruption may vary in duration from three or four days to a fortnight. Delirium at night is not unusual. Convalescence becomes established, and desquamation occurs in the course of one to three weeks. During this last stage albumen may appear in the urine, if it has not appeared before.

Diagnosis.—Erysipelas is to be diagnosed from *erythema* complicated by cellulitis, in which the margin is less raised, and there is less fever. In *herpes* of the first division of the fifth nerve vesicles occur in groups, are limited to one side of the face, and are unattended by fever.

Varieties.— (i.) Phlegmonous erysipelas or gangrenous erysipelas are severe varieties with suppuration or extensive sloughing. (ii.) Erysipelas neonatorum is a very fatal variety ; death may be due to peritonitis by inflammation spreading along the umbilical cord. (iii.) Erysipelas of the fauces is a severe variety, the eruption spreading to, or starting in, this situation. The disease may spread to the larynx and cause fatal dyspnœa.

Etiology.—It is a highly contagious malady. Persons are predisposed to it, especially alcoholics, by wounds and unhygienic conditions. It seems possible that even in so-called idiopathic cases the virus is introduced into the system through a minute and hardly visible scratch. The presence of a wound is the strongest predisposing cause, and it spreads amongst surgical patients with great rapidity. As regards age, infants

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[1] This is not usually mentioned as characteristic of erysipelas, and the first case I was called to I mistook for small-pox on this account. I have never met with a case in which it was absent, excepting in second or third attacks of the disease.

and persons over forty are most liable. The disease is due to a variety of streptococcus. One attack gives no immunity; on the contrary, it predisposes, and some elderly people are liable to an attack of facial erysipelas every year.

Prognosis.—The *usual course* is favourable, but the disease is dangerous in infancy or old persons, alcoholic or plethoric patients, and those affected with chronic diseases, especially nephritis. Death may occur by coma or syncope, preceded by incessant vomiting; or by the supervention of complications. Hyperpyrexia, persistent vomiting, lividity of the rash, and typhoid delirium are untoward symptoms.

Complications.— (i.) Subcutaneous abscesses either on the scalp, or in the neck, or elsewhere; (ii.) diffuse cellulitis, ending often in extensive sloughing; (iii.) acute œdema of the glottis from the extension of the eruption (a very serious complication); (iv.) hypostatic congestion of the lungs (very common), bronchitis, lobular pneumonia, pleurisy; (v.) peritonitis, especially when it occurs after parturition, and gastro-enteritis; and (vi.) nephritis, acute or chronic, though it is not so common as after scarlet fever - are some of the commoner complications. (vii.) Meningitis used to be mentioned as a frequent complication on account of the frequency of cerebral symptoms in erysipelas; but meningitis does occasionally occur. (viii.) Chronic ulceration or skin eruptions often disappear after an attack of erysipelas near them, and even sarcomata have been known to disappear as the result of an attack of erysipelas. This has happened even in the case of ulcers which have been of a malignant character. (ix.) Pyæmia and ulcer of the cornea are among the sequelæ.

Treatment (Hygienic Treatment, see §§ 419 *et seq.*).- A mild aperient should be given when the eruption comes out, and this should be followed by iron in large and frequent doses - 20 minims (1·2) of the liquor ferri perchloridi every four hours. Ammonia and bark are sometimes given; and in Germany large doses of quinine. Warburg's tincture is useful. Tannin or liquor ferri perchloridi are sometimes applied locally for the pharyngitis. The benefit of the latter, however, is very doubtful. *Local Treatment.*—Antiseptics, or a dusting powder of starch and zinc oxide, and the part afterwards covered with cotton-wool, or a lotion of acetate of lead and extract of opium (4 grains of each to the ounce), should be applied to the inflamed area. The eruption may sometimes be stopped by a subcutaneous injection of carbolic lotion, 1 in 20, along the margin. Some say it may be stopped by painting the advancing edge with nitrate of silver or ichthyol. Daily inspection must be made for abscess whenever the skin is tense; scarification relieves the tension, and may prevent the occurrence of suppuration. *Immunisation* is now obtainable by the use of antistreptococcus serum, and cases have been cured in this way (§ 416). The serum of convalescents has proved useful in some cases. In recurrent cases benefit is said to have been obtained by the use of an autogenous vaccine.

§ **382.** IV. **Small-pox** (Variola) is a highly contagious eruptive fever,

the eruption passing through the stages of papule, vesicle, pustule, and scab. In small-pox UNMODIFIED BY VACCINATION the symptoms are as follows :' (1) After a very definite period of incubation of twelve days, characteristic constitutional symptoms occur—viz., sudden advent of high fever (101° to 104° F.), with severe headache and *pain in the back.* The most noticeable features of this primary fever are the severity of the pain in the back (which, in my experience,[1] is present even in the mildest cases), and the frequent occurrence of vomiting. During the stage of primary fever there is, as a rule, no eruption, but in some cases a prodromal rash makes its appearance. This may be (i.) erythematous, generally found in the groins or other folds, occasionally it covers the whole body, in which case the outlook is very grave ; (ii.) morbilliform, usually occupying
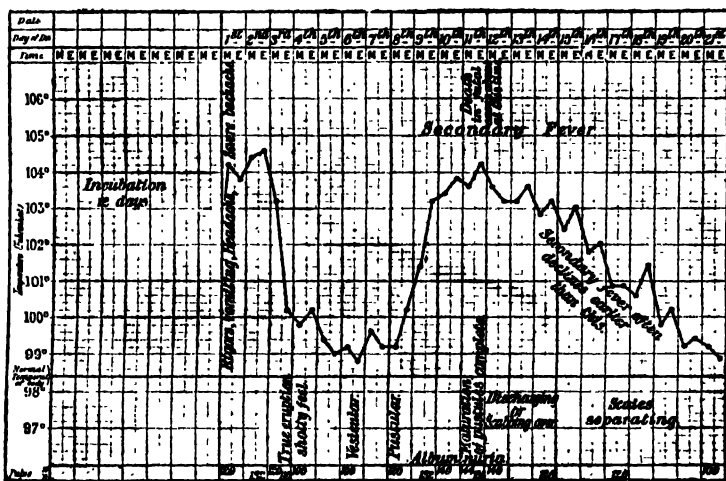
FIG. 91.—UNMODIFIED SMALL-POX.—Severe confluent case, unvaccinated, terminating in recovery. The various incidents are shown in the chart, for which the author is indebted to Dr. F. F. Caiger.

the apron area, but also occasionally diffuse ; or (iii.) a hæmorrhagic eruption sometimes appears on the anterior surface of the abdomen and thighs. Prodromal rashes appear about the second day. The fever remains up until the third day, when the true small-pox eruption appears. It then drops considerably—the patient, indeed, may feel comparatively well. About the seventh or eighth day, when the spots become pustular, a secondary suppurative fever develops, which may be attended by rigors (Fig. 91). This secondary fever lasts six or eight days. (2) The eruption appears between the third and fourth day after the illness has commenced fourteen days after infection), first as a crop of papules of *shotty hardness,* which can be felt even more readily than they can be seen, like small shot beneath the skin (Coloured Plate I). They first appear on the face

[1] Report on the Warrington Small-pox Epidemic, by Dr. T. D. Savill ; Blue Book of the Royal Commission on Vaccination. Eyre and Spottiswoode, London, 1895.

PLATE I.

VARIOLA.

Right side of face (left of observer) represents the second day of the eruption. The other, pustular, side represents the sixth day of the eruption; a few of the pustules, showing commencing umbilication.

Drawn from nature by Miss Mabel Green.

and on the fronts of the wrists, and then the eruption travels downwards
over the whole body, the abdomen, groin, and legs being least affected.
The rash may occur in the mouth, pharynx, and larynx.   Two days later
the papules become vesicular.   The eruption comes out in one crop, and
is therefore never multiform in any given area of skin, as it is in varicella.
Some of the papules, however, may abort and not proceed to vesiculation.
Each vesicle enlarges, and by the sixth or seventh day has become pustular,
presenting in typical cases, unmodified by vaccination, a depressed centre
which is held down by a bridle, a feature known as umbilication.   The
next day (eighth day) the bridle ruptures, and each pustule becomes hemi-
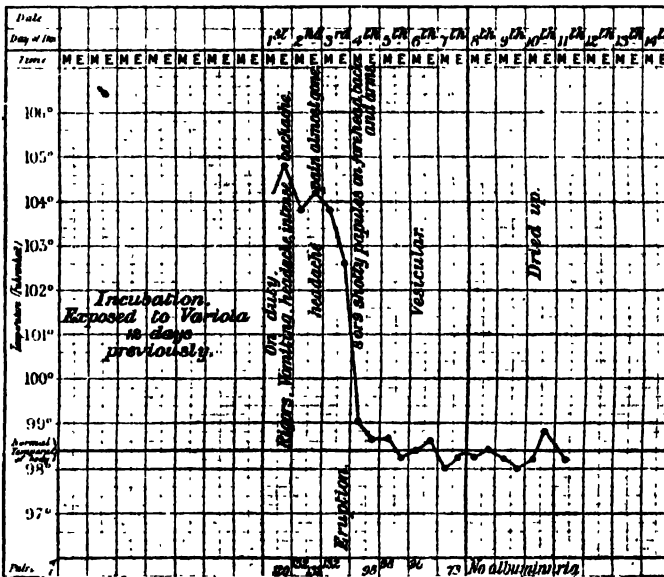
Fig. 92.—A mild case of MODIFIED VARIOLA occurring in a young woman, æt. 22, who had been vac-
cinated two years previously and who presented three visible cicatrices of the primary vaccina-
tion.   Initial symptoms severe.   No secondary fever.   The author is indebted for this chart
to Dr. F. F. Caiger.

spherical, about as large as a split pea, with an inflamed and indurated
base, and at this time considerable œdema of the skin is present.   These
pustules gradually dry into scabs, which separate about the fifteenth to
the twentieth day, though in some situations, such as the scalp, forehead,
and sides of the nose, considerably later, leaving patches of congested
skin, and in severe cases a pitted cicatrix.   The extent of the eruption
and the amount of inflammatory induration varies considerably.   Some-
times only the face and wrists present a few spots ; sometimes the whole
body is covered.   The eruption is always most profuse where the skin
has been irritated by any cause.   The eruption on the legs always presents
a proportionate retardation of development, since it appears last in this
situation.   Consequently, before certifying a patient as free from infection,

the soles of the feet should be carefully examined, and should the thick epidermis be found to harbour any dried-up remnants of obsolescent pocks, these should be carefully dug out and removed before the case can be regarded as free from possible infection.

MODIFIED SMALL-POX, or VARIOLOID (Fig. 92), is the term applied to the disease when modified by previous vaccination or natural immunity. The primary fever and early symptoms are indistinguishable from the unmodified form above described, and the eruption appears on the third day. Modified differs from unmodified small-pox in five ways: (i.) There is little if any secondary (suppurative) fever; (ii.) certain portions of the eruption abort and do not pass through all stages; (iii.) as a consequence, several stages of the eruption may occasionally be seen on the same portion of skin; (iv.) the individual lesions are smaller or more superficial than usual; (v.) the
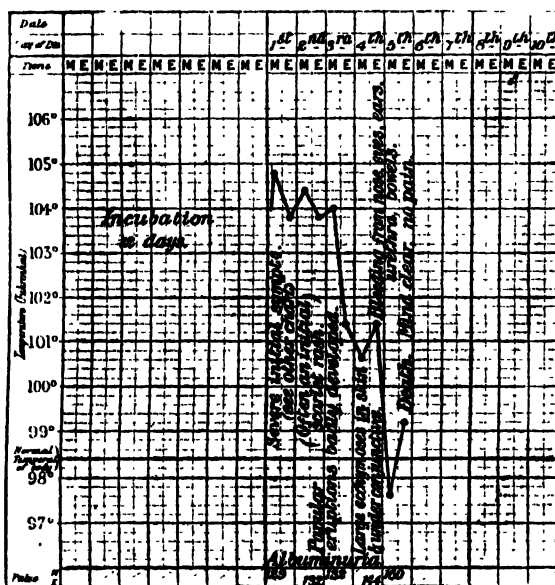
FIG. 93.—A case of MALIGNANT HÆMORRHAGIC SMALL-POX (as distinct from those cases of confluent small-pox with hæmorrhages in the pustules).—Patient unvaccinated. Death occurred on the 5th day. The various incidents are shown on the chart, for which the author is indebted to Dr. F. F. Caiger.

general eruption may be very scanty, and may consist of not more than a dozen papules, which may not even undergo vesiculation; and (vi.) the constitutional symptoms are less severe.

Varieties.—It is sufficient to describe three varieties, according to the severity of the disease, the severity of the symptoms corresponding very closely with the character and extent of the eruption : (1) *Mild or Discrete*, (2) *Confluent*, and (3) *Malignant or Hæmorrhagic*. This form is very severe, but, fortunately, not very common. In *Malignant* small-pox there are hæmorrhages into and beneath the skin, and from most, if not all of the mucous membranes, and death ensues early (Fig. 93).

Diagnosis.—There are three important diagnostic features : (i.) Sudden

advent of high fever, often accompanied by a rigor ; (ii.) headache, *backache*, and vomiting at onset of the disease, of which there should always be a history, even in the mildest cases ; and (iii.) the shotty character of the papules.[1] *Measles* is the disease which is most often mistaken for variola in the early stages of the case, and therefore two plates of these diseases are presented side by side (Coloured Plates I and II). Measles is distinguished by the redness of and the running from the eyes, with other signs of catarrh; and the presence of Koplik's spots on the buccal mucous membrane. The rash, too, is macular rather than papular, and the individual spots as they increase in size spread out in patchy coalescence. *Varicella* is distinguished by the inappreciable character of the premonitory constitutional symptoms ; by the centripetal distribution of the rash and more superficial character of the individual lesions ; by the fact that the temperature rises as the rash appears, instead of quickly subsiding ; by the eruption coming out in a succession of crops, so that one portion of skin may show several stages of the eruption ; by the rapidity with which the rash passes through the successive stages of development ; and by the vesicles not being preceded or accompanied by any *shotty induration.* In febrile *roseola* or lichen, the fever lasts only twenty-four hours, the efflorescence appears all over the body at once, and it does not go to any further stage. *Pustular syphilide* is chronic, and is unattended by any marked pyrexia.

Etiology.—The malady is highly infectious, but its specific cause has not yet been discovered. Guarnieri has described a protozoon as constantly present in the epithelial cells of the small-pox vesicle, and this is supported by Councilman. Its causal rôle, however, has not been so far established. Children, and especially infants, are particularly prone to the disease, and before the discovery of vaccination (A.D. 1796) it was a cause of considerably more than half the infantile mortality in Great Britain and other countries.[2] The poison is conveyed through the air to a considerable distance. Some believe it may be conveyed to a distance of miles, but this is very doubtful.[3] One attack confers complete immunity in most instances ; authenticated second attacks are extremely rare.

[1] See also a paper by the author " On the Diagnosis of the Early Stage of Small-pox," *Brit. Med. Journ.*, April 29, 1897.

[2] It is a fact of some interest that Warrington was the scene of an epidemic of small-pox in 1773, and the death-rate from the disease in that year was 26·5 per 1000 (211 deaths, and, reckoning five inhabitants to a house, 8000 inhabitants), all the deaths occurring in persons under nine years of age.—Dr. Thomas Percival, F.R.S., Phil. Trans., 1774, vol. lxiv.

In 1892–1893 Warrington was again visited by an epidemic, and the death-rate was then 1·1 per 1000 of the inhabitants, who had at that time only about 1 per cent. unvaccinated persons among them.

[3] This question has been very hotly debated, but in the author's belief there are no definite evidences of small-pox being conveyed through the air to a greater distance than a few yards. It is extremely contagious, and all the cases supposed to be due to aerial spread can, if sufficient information can be procured, be explained by the conveyance of contagion either from person to person, or through some mediate agency. —Report on the Warrington Small-pox Epidemic, 1892–1893, pp. 64–77. Appendix to the Report of the Roy. Com. on Vaccination.

Prognosis.—Vaccination.--The case-mortality of small-pox in the present day is about 37 per cent. amongst the *unvaccinated;* about 5 or 6 per cent. among all classes of the *vaccinated* taken together ; and about ¼ per cent. among the *properly vaccinated.* The severity of the disease seems to depend almost entirely upon whether the patient has been recently and efficiently vaccinated.[1] In the healthy and recently vaccinated it is a comparatively trivial disorder, but in the unvaccinated, especially in infancy, it is one of the gravest diseases. The second factor in the prognosis is the question of *age;* and the official records of the unmodified outbreak in Warrington in 1773 show that of 211 fatal cases 166 were under three years of age. Alcoholism and plethora add to the gravity of the disease. The greatest danger is on about the eleventh day in the confluent form. As regards the *varieties,* the confluent, in which the rash may come out on the second day, and is very abundant, is much more dangerous than the discrete form. In the former the fever does not subside on the third day, and there is a great tendency to hyper-pyrexia and complications. Speaking generally, the more copious the rash, the greater the danger. True hæmorrhagic small-pox is invariably fatal, but if hæmorrhage occurs *into* the vesicular or pustular rash, there is a good chance of recovery. As regards untoward *symptoms,* the more severe the primary fever in the unvaccinated, the more severe will be the disease, but this is not necessarily so in the vaccinated ; profuse salivation is a bad symptom ; the case is grave if there be no swelling of the skin at about the ninth day, and still graver if the swelling goes suddenly away ; convulsions and other complications are unfavourable.

Complications.— (i.) Acute laryngitis or œdema glottidis is an occasional cause of death. Hypostatic congestion, pleurisy, empyema, erysipelas, and pneumonia are apt to occur. (ii.) The heart may be affected with peri- or endo-carditis ; but myocarditis and granular degeneration are more common ; (iii.) nervous complications, *e.g.,* encephalitis, hemoplegia, acute ataxia and post-febrile psychoses ; (iv.) ophthalmia and consequent destruction of the eye is common in the East ; painless corneal ulcers may form and perforate ; (v.) cutaneous abscesses are very common during the period of desiccation.

Treatment.— It should be remembered that vaccination is capable of modifying the disease even after exposure to infection, because the incubation period of variola is twelve days and that of vaccinia only eight days. Vaccination may, therefore, be performed with efficacy during the

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[1] The figures from the Warrington epidemic, 1892–1893, are very striking. In the *infected* houses there were 2535 persons, and 2223 of these persons had been vaccinated in infancy. Among these latter 521 (23·4 per cent.) were attacked, and 27 died, so that the case-mortality among them was 5·2 per cent. There were in the *infected* houses 107 unvaccinated persons, of whom 60 (56·1 per cent.) were attacked, and 21 died, giving a case-mortality of 35·0 per cent. The figures also showed that in proportion as the vaccination had been more efficient, the severity of the disease was less. Finally, among all the 667 cases which occurred in this epidemic, not one had been vaccinated or revaccinated within seven years of the attack.—Appendix to the Report of the Roy. Com. on Vaccination, 1894.

first three or four days after exposure ; and every member of an infected household should be vaccinated immediately the disease breaks out therein. As regards *therapeutic agents*, little is necessary in the Discrete form beyond a mild aperient and salines. In the Confluent form stimulants are necessary, and we must watch for complications, and meet them as they arise. To this effect the eyes should be examined in a good light daily. If much salivation be present, it may lead to suffocation. The patient should be put into a warm bath and kept there for a considerable time. For sore throat use gargles, syringing, or swabbing ; for œdema glottidis, inhalations, or tracheotomy may be necessary. Many devices have been contrived to *prevent scarring* by the eruptions, such as powdering with zinc and starch powder, or with pulv. cretæ aromaticus, with a small quantity of disinfectant, or laying on lint soaked in glycerine and water, with a drop or two of carbolic acid, or painting the whole body with a 5 per cent. solution of permanganate of potash. Another method promised to be efficacious--namely, placing the patient in a room from which all but the red rays of the spectrum are excluded by pasting red paper over the windows. The red light treatment has been reported on very favourably by Finsen, but has not proved very successful in this country. *Hygienic Treatment* is given in §§ 419 *et seq.*

The *Preventive Treatment* of small-pox is accomplished in the present day by three means--disinfection, isolation, and vaccination. Concerning the first two see § 419 ; **for** evidence of the efficacy of vaccination in the prevention and modification of small-pox, see p. 522 and below. *Inoculation* used to be practised because it was found that the inoculated disease was milder, and usually gave as much immunity from a second attack. Out of 20,000 inoculated by the brothers Sutton not one died. It was, however, declared illegal in 1840, as it tended to spread infection.

§ 383. **Vaccinia.**--VACCINATION is the production in a person of the disease called vaccinia, by inoculating him with the lymph taken from the udder of a cow or calf suffering from that disease. It was noticed in 1769 by a German that people engaged in the milking of cows were exempt from small-pox. Jenner, in 1796, placed the subject on a scientific basis, and ascertained that the inoculation of a human being with the lymph taken from the unbroken vesicles on the udder of a calf suffering from vaccinia protected that person from small-pox. He was also the first to inoculate this disease (vaccinia) from person to person by taking the lymph from the vesicle on the arm which had matured on the eighth day after inoculation. Vaccination was made compulsory in 1853. In 1897 this law was repealed in response to an outcry among the public that syphilis and (?) other diseases could be conveyed from person to person in this way. Syphilis certainly has, in rare instances, been conveyed by arm to arm vaccination ; but by using calf-lymph this is entirely obviated ; and all public vaccinators now use lymph direct from the calf. Anyone who now goes before a magistrate and solemnly declares that he has " conscientious objections " to vaccination can procure

exemption for himself and his children from compulsory vaccination.

Rules for vaccination. - Calf-lymph is now universally used in Great Britain.[1] The best method is that of scraping the cuticle on the inner and posterior side of the arm with a blunt-pointed lancet or needle. A subcutaneous method has been recommended to avoid the risk of secondary infection and the presence of a scar. Congenital debility and an extensive skin eruption, which may give rise to generalised vaccinia, are the only contra-indications to vaccination.

The Phenomena of Vaccination. - There are no symptoms for the first two days. On the second or third day a slight pimple, on the fifth day a bluish-white cupped vesicle appears, and on the eighth day (the same day of the week as that on which the operation was performed) the vesicle *becomes matured*. It should never become purulent, but the areola increases during the next two days. The contents then become cloudy, and after the tenth day they dry up ; the scab falls ôn the fourteenth or fifteenth day, leaving a pitted cicatrix. In revaccination the reaction usually appears earlier and the vesicle becomes mature sooner than in primary vaccination.

The inquiries which the author made on behalf of the Royal Commission on Vaccination into the Warrington Epidemic (*loc. cit.*) went to prove (1) that efficient primary vaccination offers absolute protection against *infection* for the ensuing five or six years, and relative protection (gradually diminishing) for a considerable time ; (2) that primary vaccination lessens the *severity of the attack* of small-pox if contracted during the ensuing twenty or thirty years ; (3) that revaccination affords absolute *immunity from attack* during the ensuing five or six years, and relative protection for the rest of life ; and (4) that if everybody were vaccinated in infancy and again at twelve and twenty-one, small-pox would be exterminated.

§ **384.** V. **Measles** may be defined as an infectious febrile disease attended by catarrh of the ocular, nasal, and respiratory mucous membranes, and by an eruption of minute elevated papules which, as they enlarge, become aggregated into irregular and often crescentic groups.

Symptoms. - (1) After an incubation period of seven to fourteen days, usually ten or eleven, the pyrexia (Fig. 94) comes on abruptly, though not so suddenly as in scarlet fever, rising to 102° or 103° F. on the evening of the first day. The next day or the day after it usually declines a little. When the rash appears on the fourth day it rises again, remains up until the sixth day, and then falls by crisis. (2) The fever is attended by symptoms of coryza for which, indeed, the case may be mistaken if the temperature be not very high. There are profuse lacrymation, œdema of the palpebral conjunctiva, sneezing, running of the nose, and bronchial catarrh, the larynx and bronchi being specially involved. The fauces

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[1] If human lymph is employed, it should be taken from a child, not an adult, and the child should be in good health, and free from any evidences or history of syphilis. The lymph should be taken from a vesicle before it becomes opaque, and before the areola has formed.

PLATE II.

MEASLES.

The eruption, which is very plentiful, is eighteen hours old (second day of rash).
Note the evidence of coryza in the eyes and nose

Drawn from nature by Miss Mabel Green.

ar\sore, and mottled with redness, but not much swollen. (3) Koplik has described spots, which appear from one to three days before the skin rash on the buccal mucous membrane opposite the bicuspid or molar teeth, and just within the angle of the mouth. They are not easy to see, and require a good light, but they may be very numerous, when they give the appearance of a white stippling on a slightly raised reddened base. They occur in more than 90 per cent. of all cases, and, consequently, lend great help to the diagnosis in the early stages. (4) Prodromal rashes [1] in the form of isolated macules, blotchy or scarlatiniform erythema, and urticaria may appear during the first three days. The *eruption* appears
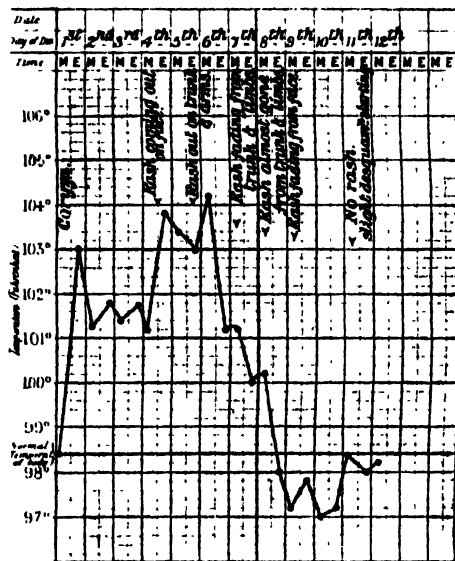
Fig. 94.—MEASLES.—Ethel H——, æt. 5 (under the author's care). Typical chart. The various incidents are shown upon the chart.

on the third or fourth day (Coloured Plate II). It consists of red, raised, well-defined flat papules, discrete at first, but afterwards tending to coalesce into irregular-shaped patches. The colour is a reddish-brown, disappearing on pressure. The spots first appear on the *face* behind the ears and side of the neck, where they are most abundant, and then pass downwards. Each papule reaches its maximum in about twelve hours to twenty-four, and then feels soft and velvety, thus differing from the early stage of small-pox papules. They soon begin to recede, and at the end of forty-eight hours to fade. By the eighth or ninth day the eruption of measles has completely disappeared, except that a brownish mottling of the skin may remain for some time after. Occasionally the macules become

[1] See paper by J. D. Rolleston on "The Prodromal Rashes of Measles," *Brit. Med. Journ.*, 1905, i, 233.

petechial. Sometimes the eruption suddenly disappears—the result, probably, of some internal complication. The catarrh goes on increasing during the development of the rash, and they subside together about the sixth to the eighth day, when convalescence commences. Slight desquamation of minute branlike scales, chiefly on the face, neck, and arms, occurs sometimes. The blood shows a characteristic leucopenia which is replaced by leucocytosis on the occurrence of a complication.

The *Varieties* are less well defined than in scarlatina. The malignant or hæmorrhagic variety (*toxic*), now, fortunately, rare, is very severe, and is attended by petechiæ and the typhoid state. The rash or catarrh may be absent in exceptional cases. In the *Pulmonary* variety, bronchopneumonia seems to be present from the commencement of the attack.

The *Diagnosis* from a severe " catarrh," in the absence of Koplik's spots, is very difficult until the eruption appears. *Variola* often presents a difficulty, though the absence of catarrh, and the presence of pain in the back and vomiting, aid us considerably in diagnosing variola. The differences between the rashes are referred to above. *Erythema Multiforme* is somewhat like measles, but is recognised by the absence of catarrh, pyrexia, and of Koplik's spots. That set up by the injection of an anti-serum is especially suggestive, and may lead to temporary confusion. The paramount importance of Koplik's spots in the early diagnosis of measles can hardly be exaggerated.

Etiology. Measles is especially a disease of childhood, and few escape. It is endemic in England, and outbreaks occur from time to time. The seasonal prevalence is in the spring and winter. The essential cause is probably a living organism, which has not yet been identified. It is conveyed chiefly by the breath and nasal mucus. Caronia (*Presse Méd.*, 1923, XXXI, 877) regards a minute diplococcus which he has isolated on special media from the blood, bone-marrow, naso-pharyngeal secretion and cerebrospinal fluid, during the prodromal and eruptive periods, as the causal organism of measles. Unlike scarlatina, it is as contagious before as after the eruption has appeared, and its infectivity disappears more rapidly. One attack confers relative immunity ; second attacks are less common than in scarlatina, the majority of so-called second attacks being probably Rubella.

Prognosis.—Measles is not as a rule a serious disease in itself, except in infancy. The case-mortality in an outbreak does not often exceed 2 or 3 per cent., though it may be as high as 10 or 12. The most important determining factors are poverty and the proportion of very young children. The chief danger of the disease rests in the complications and sequelæ which may attend even the mildest case of measles. The prognosis is bad in proportion to the severity of the pyrexia and pulmonary symptoms. Strumous or weak children suffer most. Convulsions late in the disease are of grave significance. The most important and most common *complications* are bronchitis, broncho-pneumonia, pneumonia, and collapse of the lung, and diarrhœa, especially in the summer months. Phthisis •

is a recognised sequela ; it follows measles and whooping-cough more frequently than any other febrile disease. Catarrhal laryngitis, diphtheria, and laryngismus also occur. In all cases of measles with sudden aggrava- tion of fever and no apparent cause, the presence of acute otitis media may be suspected. Cancrum oris beginning as an ulcer on the internal surface of the cheek and surrounded by intense inflammation is compara- tively rare now-a-days. Soon a black slough appears, followed by per- foration. Occasionally it starts in the gums in the neighbourhood of a carious tooth, and the alveolus may be involved in the necrosis. Gangrene may occur in other parts, such as the genital organs. Other complications are ophthalmia, keratitis, stomatitis, enteritis, rhinitis and encephalitis. Caseous bronchial glands are common sequelæ.

Prophylaxis.— Subcutaneous injection of the serum of convalescents within the first six days after exposure to infection in nearly all cases either prevents an attack altogether or renders it very mild. Two injec- tions are given, with one day's interval between each, the dose being 2 c.cm. in infancy and 4–6 c.cm. in older children.

Treatment.— A hot bath may be given at the onset. Remedies are directed against the bronchitis, the most useful being ipecacuanha and liq. ammoniæ acetatis, F. 53 (General Treatment, see §§ 419 *et seq.*). The early application of a jacket poultice in the case of infants with bronchial involvement will often work wonders.

§ 385. VI. **Rubella, or German Measles** (Synonym : Röteln) may be defined as an acute contagious disease, characterised by sore throat, catarrh of the respiratory passages, and an eruption of the skin, consisting of small pinkish-red spots, which afterwards become confluent. Clinically, it may be said to represent a combination of measles and scarlatina, giving rise to a diffuse redness of the surface. Undoubtedly, many so-called cases of Rubella are identical with measles.

The *Symptoms* vary somewhat in different epidemics. (1) After a period of incubation, variously stated to be from seven days to two or three weeks, but more often ten to seventeen days, the temperature rises to 100°, 101°, or 102° F. This is accompanied by sore throat and coryza. Usually the glands are swollen, the most characteristic being the concatenate and occipital groups. Tender swelling of the posterior cervical glands is sometimes present several days before the rash appears, the patient often complaining of "stiff neck," which he usually ascribes to having sat in a draught, or some such reasonable explanation. When the eruption comes out, the other symptoms are considerably aggravated, but the whole attack rarely lasts as long as a week. The rash may be the first indication of the disease, as the primary fever is sometimes so slight, or it may be entirely absent. (2) The eruption is sometimes delayed until the third or fourth day of attack, and consists of minute round or oval rose-red spots, varying in size from a pin's head to a pea, very slightly raised, never papular. The rash at the outset is like that of early measles. In a day or two it becomes confluent, or nearly so, and the whole skin presents a scarlet hue, so that the case may be mistaken for scarlatina. The eruption first appears on the face, and at the end of twenty-four hours the whole body is involved. It lasts from two to five days, and the severity of the attack is in direct ratio to the duration and severity of the eruption. It is sometimes followed by slight desqua- mation. The disease has to be *diagnosed* from *scarlatina*, in which there is no catarrh and no "measly" eruption at the beginning of the attack, but the tongue will show the "strawberry" character. In *measles* one should look for "Koplik's spots," but there are no enlarged glands, no special involvement of the tonsils, but little sore

throat, and no extensive confluence of the rash. In·non-specific *roseola* (rose rash), there are no catarrh and no sore throat. In some *drug rashes*, especially that due to copaiba, which may simulate rubella, there is no cervical adenitis or rise of temperature.

Etiology.—It is mainly a disease of childhood, but sometimes attacks adults. It is not so contagious as either scarlatina or measles (Murchison). One attack confers immunity. The disease is spread by direct contact only and not by fomites or healthy third persons.

Prognosis.—It is a much more trivial disease than measles. Complications are very uncommon and recovery always takes place apart from intercurrent disease.

The *Treatment* calls for no special remark.

§ **386.** VII. **Dengue** is an infectious fever, of tropical and subtropical climates, which is due to the inoculation of an unknown virus by the bite of *Culex fatigans*. The incubation period of dengue is three to six days. The fever is of a sudden onset, and ranges from 102° to 105° F. It is accompanied by intense headache, with extremely severe pains in the joints or limbs, much aggravated by movement. This primary fever lasts about forty-eight hours, and subsides by crisis. At this stage the skin may be covered with a bright red flush, chiefly about the face and neck. Hæmorrhage from the nose or stomach may also occur. During the next one to two days there is an interval of apyrexia, with freedom from pain. Occasionally this interval is absent. Then the secondary fever appears, with a return of the pains in the limbs. Both, however, are less severe than in the primary stage. A universal mottling of the skin, starting on the hands, somewhat resembling measles, though never papular, may accompany the secondary fever. As it subsides in a day or two, slight branny desquamation occurs. There is leucopenia during the fever and eosinophilia in convalescence.

Diagnosis.—Dengue is known from *scarlet fever*, which is rare in the tropics, by its being rarely associated with sore throat or enlarged cervical glands, by the severe articular pains, and by its occurring in hot weather, and, later, by its characteristic temperature. *Acute rheumatism* is rare in the tropics, has no rash, and has profuse sweats. *Measles* has coryza and Koplik's spots ; *influenza* rarely has a rash.

Prognosis.—As regards life, the prognosis is excellent ; the case-mortality is extremely small. Death rarely, if ever, occurs ; if so, it is from such complications as weak heart or hyperpyrexia, in the enfeebled. In most cases the acute symptoms have passed off in eight days. Some have painful joints and crippling for some time after the fever has gone. The disease confers immunity for some little time.

The *Treatment* does not differ from the ordinary hygiene necessary in fevers. The patient should be kept in bed. For the pain in the limbs, belladonna, aspirin, antipyrin, and even morphia may be given. The subsequent anæmia and enfeeblement are sometimes troublesome. Prophylactic treatment is that for malaria, with the exception of the use of quinine.

§ **387.** VIII. **Typhus** (Synonyms : Spotted Fever, Exanthematic Typhus, Hospital, Gaol, and Ship Fever) may be defined as a contagious fever, lasting fourteen days, with an eruption on the skin consisting of subcutaneous mottling and petechial spots, with a great tendency to the typhoid state. Its disappearance from our midst is a good illustration of the triumphs of hygiene. It is probably due to *Rickettsia prowazeki*, *i.e.*, minute intracellular organisms found in the bodies of infected lice as well as in typhus lesions in the human subject. The disease is endemic in Eastern Europe, North Africa, Asia Minor, Arabia, Persia, China, Japan and Mexico.

Symptoms.—(1) After an incubation period, which varies considerably, but is rarely longer than twelve days, the temperature continues to rise during the first few days. It may then fall slightly during the second week, and usually ends by crisis about the fourteenth day or earlier. It starts somewhat abruptly with chilliness, rarely with rigors. There is severe headache and extreme prostration, so much so that on the second day the patient is unable to walk or stand. Drowsiness is common, and there is a typical aspect of heavy stupidity. At the end of the first week headache gives place to delirium, and this is followed by drowsiness and coma. (2) The spleen

is enlarged and tender.   (3) The eruption appears usually on the fourth or fifth day, first on the back of the hands, arms, folds of axillæ, and in front of the chest and abdomen.   It has usually two elements, which vary in their proportion : (a) Sub-cuticular mottling, certain portions of the skin appearing hyperæmic, with fading margins ; (b) purple, or brownish-red spots, having a definite but irregular outline, varying in size from a pin's head to three lines, very slightly elevated at first, and in the course of two or three days becoming petechial, so that they will not disappear on pressure.   One attack usually confers immunity.

Diagnosis.—The Weil-Felix reaction (*i.e.* agglutination of B. proteus X19 by the serum of typhus cases in dilutions of 1-100 to 1-2000 or higher) is a very valuable reaction, but is not obtained until the end of the first week.   (1) *Typhoid fever* was originally confused with typhus, and it is chiefly owing to the observations of Sir William Gairdner and Sir William Jenner that they are now differentiated.   Typhoid differs from typhus in (i.) the insidious onset ; (ii.) the course of the temperature ; (iii.) the different eruption ; and (iv.) the diarrhœa and pea-soup stools.   (2) In *measles* the eruption resembles the typhus spots, and appears at the same date, but in typhus it does not involve the face, it is never preceded by catarrh, is never papular, and becomes petechial.   (3) Some *malarial* fevers present considerable difficulty, but they have no eruption.   (4) *Uræmia* and other causes of coma may be mistaken for it.   (5) *Pneumonia*, meningitis, and other causes of the *typhoid state* may be confused with typhus.   (6) Epidemics of *plague* have been confused with typhus, but the parotid swellings in plague occur earlier, during the first week.

Etiology.—The disease is met with at all ages, but is more dangerous in middle and advanced life.   In children it is usually a mild disease and often not recognised.   Doctor and nurses frequently contract it ; Dr. Charles Murchison contracted typhus twice, and thus incurred the heart disease of which he died.   The disease is associated with overcrowding and personal squalor.   Nicolle, Sergent and Foley have proved that the disease is due to a filterable virus which is conveyed by lice.   Recent experience of the epidemic in Serbia (1915) confirms the conveyance of the virus by body lice.   The malady is predisposed to by a general debility, and it is therefore commoner in times of famine and distress.   It is most prevalent in winter and spring.

Prognosis.—Case-mortality, 10 per cent. : between the age of fifteen and twenty-five, 4 per cent. ; over fifty, 50 per cent. or more.   Thus the age of the patient greatly influences the mortality.   Typhus is always a serious disease, especially in the plethoric and alcoholic.   It terminates fatally in three ways : (i.) Degeneration of the cardiac muscle, which is a very common accompaniment of the disease ; (ii.) coma, from the toxic state of the blood ; or (iii.) asphyxia or hypostatic congestion of the lungs.   Untoward symptoms are (i.) weak, irregular, or intermittent pulse, or other indications of cardiac weakness ; (ii.) an abundant rash, with high fever ; (iii.) early and protracted cerebral signs or protracted hiccough ; (iv.) all complications, especially pulmonary.   Of the *complications* and *sequelæ*, (i.) the pulmonary are the worst, especially broncho-pneumonia and hypostatic congestion of the lungs ; œdema glottidis and pleurisy are less common.   Other complications are (ii.) hyperpyrexia and meningitis ; (iii.) femoral and other thromboses ; (iv.) gangrene of the extremities from embolism, bed-sores and pyæmic abscesses ; (v.) cardiac weakness, which may remain for a long time, on account of the granular degeneration of the muscle ; (vi.) post-febrile mania ; and (vii.) paralysis of various parts.

Treatment.—Prophylaxis consists in strict avoidance of lice.   Deterrent powders should be used on the body, and protection from the insect is attained by overalls fitting tightly round neck, wrists and ankles.   Hygienic treatment is essential (§§ 419 et seq.), especially free ventilation.   Cases treated in the open air do best.   Therapeutic treatment is chiefly symptomatic, and for this reason strychnine is useful.   Stimulants in most cases are not necessary, but they must be given if the pulse is weak or irregular, or if the extremities are cold.   Treatment by serums and vaccines is still in the experimental stage.

§ 388. Anthrax, or Malignant Pustule (Synonyms : Woolsorters' Disease, Anthrac-

æmia, Splenic Fever—under which term the disease is registered in the Registrar-General's returns—Charbon, Carbunculus Verus). The primary lesion consists of a solitary vesicle at the seat of inoculation. As the base of this becomes transformed into a central slough, the contents become hardened, and around this a zone of vesicles arises. Pasteur showed that it is due to the anthrax bacillus, a relatively large organism which was one of the first to be isolated.

This disease, which has a marked and prolonged vesicular stage, is most usually situated on the dorsum of the hand or arm, occasionally on the face ; 82 per cent. of the cases show pustule on the head or neck. It affects woolsorters, furriers, felt-makers, ragsorters, and others who come in contact with animals or their hides or fur ; 40 per cent. of the cases in British leather-workers are due to handling Chinese or East India goods. No case has been traced to wet-salted hides. The incubation period is twenty-four to seventy-two hours. First a papule forms at the seat of inoculation, which rapidly enlarges, and becomes on the second day a vesicle, with serous or hæmorrhagic contents. On the third day this bursts, leaving a raw exuding surface, which, on the fourth day, turns to a dry black slough, surrounded by a zone of intense inflammation slightly raised above the surface. Upon this inflammatory zone there appears, also on the fourth day, a characteristic ring of small red vesicles. The œdema extends around, and the lymphatics and the glands inflame. The pain is usually very slight, and no pus forms until about the tenth day, when the slough begins to separate. The constitutional symptoms vary considerably, and bear no proportion to the local mischief. The pyrexia may be so slight as not to interfere with the patient's ordinary avocation, and it may not come on until some days after the local signs. Usually, however, it is severe, comes on early, and soon assumes a typhoid character.

Intestinal and *Pulmonary* types are also described, according to the method of infection. In the former intense vomiting and diarrhœa occur, with great prostration and cramps, with, in some cases, cyanosis and dyspnœa, and towards the end convulsions and spasms. The spleen is enlarged. In the latter, which is caused by inhalation of diseased wool or hair (*woolsorters' disease*), there are urgent dyspnœa, and pain in the chest of sudden onset. The temperature rises to 102° or 103° F., and death may occur with profound collapse in twenty-four hours. Sometimes delirium and convulsions, or diarrhœa and vomiting, occur.

Diagnosis.—It may have to be diagnosed in the first place from the sting of an insect, from various conditions which lead to solitary vesicles or bullæ on the second day, from erysipelas (if on the face), lymphangitis, and other cases of œdema. The occupation of the patient assists us, but a diagnosis may be made by examining the serum or secretion of the sore, stained by Gram's method (Chapter XX), under the microscope. The *Bacillus anthracis*, which is the cause of the disease, is thus readily discovered.

Prognosis.—The mortality varies with the position of the primary lesion, being 40 per cent. when this is situated on the neck or face, and 12 per cent. when situated elsewhere.

Treatment.—The local lesion should be freely excised if seen early, and the wound irrigated continuously with carbolic lotion (1 in 20). The lotion may also, with advantage, be injected into the tissue surrounding the part, and repeated every four hours, due care being taken to watch for carbolic poisoning (carboluria, etc.). Good results have been obtained by injection of neosalvarsan. Sclavo has prepared an anti-anthrax serum, which should be given locally into the indurated border of the lesion in addition to general administration. Recent reports (1924) as to its value have been very favourable.

§ 389. Glanders (Synonym : Equinia) may be defined as a contagious febrile disease attended by a discharge from the nostrils, and sometimes an eruption on the skin, due to the inoculation of the *Bacillus mallei*, in a person attending to HORSES affected with the disease. The eruption, which only occurs in ACUTE GLANDERS, consists of a general erythema, on which a crop of pustules of hemispherical shape appear in the

course of a few days or hours. They vary in size between a lentil and a florin. There are also nodules of granulomatous material in the subcutaneous tissue and muscles, which usually suppurate, leaving large foul ulcers. The other symptoms are (i.) a copious discharge of viscid, semipurulent matter from the nostrils ; (ii.) pains in the limbs and joints ; and (iii.) high fever, with rigors and prostration, passing on to the typhoid state.

In CHRONIC GLANDERS (Farcy) the pyrexia and constitutional symptoms are absent, and the cutaneous eruptions (erythema, pustules, and nodules which leave ulcers and sinuses). The discharge from the nose may be the only sign.

Diagnosis.—The pustules of acute glanders resemble those of variola, but they are larger, and not umbilicated, and the temperature in glanders does not fall when the rash—in those cases which present a generalised pustular eruption—comes out.[1] The pain and swelling of the joints and limbs bear some resemblance to acute rheumatism, and still more to pyæmia. The reaction to mallein may assist.

Treatment.—Vaccination with small doses of dead bacilli is advocated, and has been tried in a few cases. At present the disease is extremely fatal. In FARCY or CHRONIC GLANDERS the death-rate is 40 or 50 per cent. Iodide of potassium, aconite, mercury, iron, arsenic, and strychnine have all been tried, and good results have accrued from the injection of small doses of mallein.

GROUP II. CONTINUED PYREXIA

§ 390. In this group the pyrexia tends to assume a CONTINUED TYPE —*i.e.*, it runs a continuous course except for the slight normal diurnal variation (§ 374). This group is distinguished from Group I by the absence of an eruption during the first four days of the illness. It is distinguished from Group III mainly by the course of the pyrexia, though aberrant types of one group are found in the other.

Rocky Mountain Fever and some of the other fevers rare in this country have an eruption which develops usually after the fourth day.

TYPHOID FEVER, which may be taken as a type, may in exceptional cases present no other symptoms than *the characteristic pyrexia.* The rash, when present, may be ill-marked, and does not appear till the second week of the disease. In DIPHTHERIA there is the characteristic *throat lesion ;* in INFLUENZA there are *pains in the limbs*

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[1] The author once notified a case of this kind as small-pox, and the case passed as such through the hands of two of the most experienced medical officers of the Metropolitan Asylums Board, the mistake not being cleared up until after death, and a full investigation had been made of the *circumstances under which the disease arose.* It was then ascertained that the patient was a stableman, attending on glandrous horses.

and a more sudden advent ; in PERTUSSIS the *characteristic cough ;* and in MUMPS the *parotitis.* Various MICROBIC REACTIONS may aid us in the diagnosis. CHOLERA (§ 245) and DYSENTERY (§ 244) might also be included in this group, but the pyrexial disturbance is quite a subordinate feature compared with the intestinal manifestations. Dr. Cabot (*loc. cit.*) analysed 784 cases of fever lasting two weeks or longer without dropping to normal, and found that 90 per cent. were cases of enteric fever (586), sepsis (70), or tuberculosis (54). Under "sepsis" he included all forms of septic contamination of the blood-stream, as by wounds, abscesses originating from the appendix, gall-bladder, genito-urinary tract, or alimentary canal or empyema (§§ 394 and 414).

§ 391. **Typhoid Fever** (syn. : enteric) may be defined as an acute specific fever of about three or four weeks' duration, with a tendency to diarrhœa and the typhoid state, often attended by successive crops of rose-coloured spots, and due to a specific microbe (the typhoid bacillus of Eberth, see Coloured Plate IV and Fig. 107). There is a characteristic ulceration of Peyer's patches, and solitary follicles in the small intestine, and in a small proportion of cases the solitary glands in the large bowel are also affected.

Symptoms.—(1) The period of incubation is usually about ten days, but it may be shorter or longer. The onset is insidious, differing in this respect from the fevers in Group I. The most important early symptom is headache, otherwise there are simple malaise and lassitude, usually with constipation, epistaxis, bronchial cough, disturbed nights, and perhaps slight abdominal pain, which is often accompanied by a temporary looseness of the bowels. The typical typhoid chart (Fig. 95) is one of the most characteristic features of the disease, and until the discovery of the Widal reaction we were largely dependent upon this for the diagnosis of the malady. In the *first* week it is "ladder-like," gradually rising with diurnal remissions until it reaches, about the end of the first week or ten days, its highest point (103° to 105° F.). During the second stage, which may last a week or more, it remains continuously high, the diurnal remissions often being no more than those which are met with in health. As the disease progresses, these daily remissions become gradually more and more marked. During defervescence, usually about the fourth week, first the morning temperature, and then the evening temperature, gradually become normal. These features are so constant as to afford a means of detecting the stage which a case has reached. Convalescence may be said to be established when the evening temperature has been normal for two successive nights, although there is a liability for a relapse to occur for ten or twelve days longer. (2) Some diarrhœa is usually present after the first week—at least, in cases of moderate severity—and the stools are of a characteristic pea-soup or yellow ochre colour. This feature is of very little value as a means of diagnosis, while a patient is on milk diet. In more than half the cases there is no diarrhœa throughout, and the bowels are confined, but these include the large proportion of mild attacks ; complete absence of diarrhœa is exceptional in cases of any severity. (3) The spleen is generally tender and enlarged throughout the disease,

being frequently palpable even without the patient taking a long breath. Tympanitic distension of the abdomen is common if the patient be injudiciously fed, especially in the second and third weeks, and there is often pain and gurgling on pressure in the right iliac fossa, though great care should be used in attempting to elicit this symptom, as the intestinal wall is thinned by disease. (4) The eruption generally commences to come out about the seventh to twelfth day (average, tenth) in successive crops [1] of small rose-coloured lenticular spots, slightly elevated, soft, and disappearing on pressure. Each spot lasts about three or four days. They are never petechial. They are chiefly met with on the abdomen, sometimes on the rest of the trunk, very rarely on the face or limbs. The number of these spots varies considerably, but they are rarely abundant. They may be very small, and thus be overlooked or mistaken for flea-
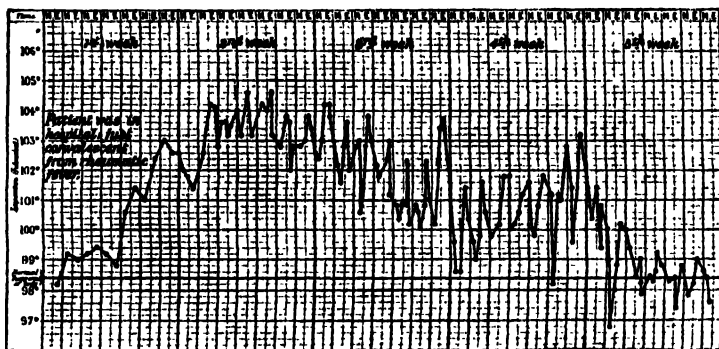
FIG. 95.—TYPHOID FEVER (typical chart), Henry H——, æt. 22 (under the author's care), was in hospital when he developed the enteric fever. There was apathetic mental condition, great feeling of illness and headache, watery pea-soup stools, and bronchial catarrh. The chart shows the continued character of the pyrexia in the second and third weeks, with gradually increasing remissions in the fourth and fifth weeks.

bites. (5) Malaise is a very constant feature from the outset, and it is for this symptom that we are generally consulted. Lethargy is very marked, and gives rise to the aspect (*facies typhosa*), which is fairly characteristic; the drowsiness deepens to semi-stupor, and in severe cases the typhoid state eventually supervenes. The tongue is first covered with a thin white fur, the edges and tip being red; in the second week the fur clears off, and the tongue becomes glazed and dry, or red and smooth. Shallow transverse fissures are often seen on it. Sordes collects on the teeth. Several *varieties* of the disease have been described, but they are not of much importance. Occasionally the disease commences quite suddenly, with symptoms of great severity. The "ambulatory" form is so called because the patient is able to keep about while suffering from it. Perforative peritonitis or intestinal haemorrhage may be its first manifestation.

[1] This fact may be revealed by enclosing each of the spots which appear on one day by a circle, next day by a triangle, and so on, by a nitrate of silver paint or aniline ink.

Diagnosis.—Until recently the diagnosis of typhoid was often a matter of excluding all other possibilities, and even then was largely a matter of conjecture. But at the present time we have a valuable test in Widal's reaction (see Chapter XX), if the patient has not been recently inoculated against typhoid fever. During the first fortnight, however, a negative Widal reaction is of no value in diagnosis. On the other hand a blood culture is most likely to be positive during the first week. The diazo reaction is positive between the fifth and tenth days and in all but severe attacks becomes negative in the second or third week. Loss or impairment of the abdominal reflex is also of some diagnostic value. Undoubtedly many slight cases of typhoid are overlooked or spoken of as *Febricula.* Slight cases are also apt to be mistaken for *Influenza,* which, except for the pulmonary symptoms, the more sudden advent, and brief duration, much resembles mild typhoid. The other *specific fevers* in this group may also have to be excluded. In most cases of typhoid there appears early in the disease a generalised *bronchial catarrh* and *hypostatic congestion of the lungs,* and nothing is commoner than to mistake enteric fever, in its early stages, for pulmonary congestion or bronchitis, and severe cases may be mistaken for *pneumonia.* These pulmonary disorders should be recognised by the relative absence of the prostration, and the diarrhœa, enlarged spleen, etc., of enteric. In severe cases of typhoid, early delirium may occur and suggest *meningitis ;* but the latter is recognised (apart from examination of the cerebrospinal fluid) by (i.) the retracted abdomen ; (ii.) the irregular and sighing respiration appearing early in the disease, and (iii.) the headache persists longer, and may concur instead of alternating with the delirium (Murchison) ; signs of intracranial pressure also supervene, such as ptosis, squint, optic neuritis, and other local paralyses. *Acute Miliary Tuberculosis* is a disease which sometimes closely resembles enteric. The positive signs of typhoid are wanting, and the presence of tubercle is suggested by (i.) the intermittent character of the temperature and its prolonged course ; (ii.) the lung symptoms are much more marked ; (iii.) the rapidity of the breathing is out of proportion to the other signs of illness ; and (iv.) the pallor and lividity of the face and the rapid emaciation are also more prominent features. *Malignant endocarditis* is recognised by (i.) the intermittent character of the temperature (usually), often with rigors, and (ii.) the cardiac signs. *Pyæmia* is differentiated by the wide range and irregularity of the pyrexia (§ 413).

Etiology.—Typhoid fever is now known to be due to a specific microbe which has been isolated. *All matters which the patient discharges from his stomach, bowels, bladder and lungs are infective.* Most epidemics are due to the contamination of the water-supply by sewage. The disease has also been traced to the eating of oysters and other shell-fish, to ice-creams, and to the milk supply. Laboratory infection sometimes occurs. To produce the malady the microbe must be introduced into the alimentary canal ; thus, nurses and friends contract the disease by handling the bed-pans and sheets, or any other articles which have been contaminated

by the fæces and urine. The excreta become more virulent after standing from twelve to twenty-four hours. The urine may contain the typhoid bacilli long after restoration to health. Such cases, as also those in which the stools have been shown to contain bacilli many years after the attack, are potent sources of infection to others, and are known as "typhoid carriers." In the latter instance the gall-bladder would appear to be the nidus from which, by means of the bile, bacilli are periodically discharged into the bowel and thus render the stools infective intermittently. The malady is most prevalent in the autumn and early winter ; and Pettenkofer has found by several years' observations that typhoid outbreaks are favoured by (i.) a rapid falling (after a rise) of ground water—that is to say, a well-aerated moist soil ; (ii.) a certain temperature of the earth ; and (iii.) pollution of the soil by animal impurities. One attack does not necessarily confer immunity, as second attacks are not very uncommon. The malady is chiefly met with in young people between ten and thirty years of age.

Prognosis.—The case-mortality varies in different epidemics from 5 to 20 per cent. The prognosis is more favourable in the young. It is always a serious disease on account of the numerous complications, prolonged course, and its exhausting nature. The usual duration is about three or four weeks, though it varies from ten days to six weeks even without relapses, which are by no means infrequent. *Untoward Symptoms.*—The height and the continued character of the fever are the best guides to the severity of the attack. Many of the fatal issues would be avoided if it were remembered that slight attacks require just as much care as severe ones, being liable to be attended by hæmorrhage and perforation if the patient does not remain at rest. The prognosis is grave when the fever remains at about 104° F. throughout the second week, and especially if the diurnal remissions do not increase, as they should do, in the third week. It is also grave when there are vomiting, except at an early stage, urgent diarrhœa at any time, severe tympanites, or hæmorrhage. A sudden fall in the temperature suggests hæmorrhage or the occurrence of peritonitis. The most common *complications* are : (1) Those of the lungs, and, as previously mentioned, bronchial catarrh and hypostatic congestion are practically symptoms of the disease. Pneumonia and pleurisy also occur. (2) Hæmorrhage, due to the ulceration of Peyer's patches, occurs in 8 or 10 per cent. of the cases. (3) Perforation. (4) Peritonitis, either local in its distribution, when it is due to the spread from the ulceration, or to perforation, is an occasional complication, and it is sometimes peculiar in being latent—that is to say, unattended by the pain which is so characteristic of that disorder. Its occurrence can then only be recognised by (i.) vomiting ; (ii.) great aggravation of the already existing prostration ; (iii.) a small rapid pulse (120 to 140) ; (iv.) immobility followed by distension of the abdominal walls ; and (v.) a sudden fall, usually followed by a rise, of the temperature ; (vi.) the *facies Hippocratica.* (5) Other complications are thrombosis of the femoral or

popliteal vein, local suppurations and inflammations, such as parotitis, periostitis, pericarditis, cholecystitis, pneumonia, pleurisy, cancrum oris, and laryngeal ulceration. As *sequelæ* multiple abscesses, various psychoses, peripheral neuritis, phthisis, and miliary tuberculosis may occur.

The temperature may rise again after convalescence has begun. Such *recrudescence* may be due to too liberal a diet, excitement, or constipation. It may, on the other hand, be due to a relapse. *Relapse* occurs in about 10 to 15 per cent. of all cases. There is usually an apyrexial interval of about five to ten days, but sometimes the temperature has never dropped satisfactorily. The second attack is usually less severe and shorter than the first, but there may be severe and even fatal relapses. As many as five relapses may occur, though more than two are rare in this country.

Treatment.—There are four indications : (*a*) to prevent peritonitis, hæmorrhage, or perforation by rest and suitable diet ; (*b*) to maintain the strength of the patient ; (*c*) to counteract the noxious effects of the specific organism, either by destroying its vitality (vaccination) or by neutralising its toxin (serum-therapy, see § 418) ; and (*d*) to prevent the development of meteorism by restricting the diet to what the patient is able to digest and, if necessary, by the administration of antiseptics. *Hygienic.*—Absolute rest is of the highest importance, and when the diagnosis has become established the patient should not be allowed to turn himself in bed. Grave responsibility rests upon the nurse in this respect, for perforation may occur in changing the draw-sheet, owing to the patient being allowed to raise himself for the purpose. It is a great mistake, however, to keep the patient continually on the flat of his back, as it not only tends to congestion of the bases of the lungs, but also conduces to bedsores. He should be encouraged to lie on his side, and should be carefully turned every two hours on either side alternately. In contradistinction to the febrile diseases already described, typhoid fever patients may be treated in a general ward, but great care must be taken to keep all utensils and the thermometer apart and disinfected. The stools must be burnt or immersed in izal, or some other reliable disinfectant, directly they are passed, and 1 in 20 carbolic must be added to the urine. All linen must be first steeped in an antiseptic such as a solution of carbolic acid, or lysol or izal, for several hours, and then boiled. All pots, pans, mugs, etc., must be boiled after each occasion on which they have been used. *Diet* is also of prime importance. Milk is the staple article, and not less than 2 and not more than 3 pints a day should be given, sufficiently diluted. It is advisable to add barley-water or lime-water to prevent the formation of large curds. The addition of 3 grains (0·2) each of sodium bicarbonate, magnesium carbonate, and sodium chloride to a cup of milk, or sodium citrate in the proportion of 2 grains (0·1) to the ounce of milk, has a like result. If milk disagrees, give whey, egg albumen, or buttermilk. Clear soup, chicken broth, and beef-tea may also be given. No solids should be taken until at least one week after the temperature is normal, unless the patient resents very much the restriction. The modern

practice, however, of giving light and easily digested solid food at an earlier date than this has a good deal to recommend it. Predigested foods are of great aid to promote assimilation, especially if the tongue be heavily furred, and pepsin is said to have quite a specific effect on the disease, though it probably acts in that way. It may be given thus : Essence of pepsin, ℥xxx. (2) ; dilute nitro-hydrochloric acid, ℥v. (0·3) ; glycerine, ad ʒi. (4). The bowels must be regulated by enemata on alternate days, if required.

Hydrotherapy.—Judging from the good results which this line of treatment has given on the Continent and in America, its use is worthy of more trial than it has hitherto received in England. It is applied in three ways ; (i.) By cold or tepid sponging ; (ii.) by the ice-pack ; and (iii.) by the bath, which is used as a routine when the temperature is over 102·2° F. At the Johns Hopkins Hospital it is usual to give a bath at 70° F. every third hour, if the temperature is above 102·5° F. The patient remains in the bath for about twenty minutes, during which he is rubbed with the hand or a suitable rubber, and is then taken out, wrapped in a dry sheet, and covered by a blanket.

Medicinal.—Internal antiseptic remedies are largely used. Such are perchloride of mercury, carbolic acid, creosote, izal, lysol, β-naphthol. These are not of much value unless given in full doses, and frequently ; when fermentation, and consequent meteorism, may be prevented entirely, otherwise treatment by drugs is chiefly symptomatic. Cinnamon oil has been strongly recommended by Dr. F. F. Caiger in 3 to 5 minim doses every two hours throughout the illness. The oil must be pure, and given in capsule. If profuse, the *diarrhœa* must be checked by enemata of starch and opium (ʒss. (2) of tinct. opii to ℥iii. (12) of mucilage of starch) ; or liq. morphinæ, ℥xx. (1·2) with dilute sulphuric acid, ℥x. (0·6), every three or four hours. If this fail, give acetate of lead, bismuth carbonate, or bismuth salicylate. If *perforation* occurs, laparotomy and suture of the bowel should be performed immediately. For *local peritonitis* apply heat to the abdomen, give opium in large and frequent doses and iced milk. If the abdomen is *tympanitic*, reduce the amount of milk, or give it peptonised, or more diluted, apply turpentine fomentations and give turpentine internally. *Hœmorrhage* should be checked by the administration of opium, and absolute rest must be enjoined, and the amount of fluid given should be cut down to the smallest possible quantity. To maintain the strength, *stimulants* are called for in certain cases, but they should not be given as a matter of routine. The *pulse is the best indication* for their administration ; they must be stopped if hæmorrhage occur.

Prophylactic Treatment is based upon a knowledge of the origin of the disease and the mode of its introduction into the system—viz., by the mouth (see § 420). The incidence of typhoid in a community is a fair index of the purity of its water-supply. Preventive inoculation by means of sterile cultures of the typhoid bacillus on the lines originally recommended by Sir Almroth Wright is now an established success, notably in

the army on foreign service, while as a curative measure Chantemesse in
Paris, by means of an anti-serum, claims to have treated 1000 consecutive
cases of typhoid fever with a mortality of less than 5 per cent. Vaccine
treatment in the early stage of mild or moderate cases appears to shorten
the duration of the disease.

Paratyphoid fever, due to infection by B. paratyphosus A, B or C cannot
usually be distinguished from typhoid fever except by bacteriological
examination. Before the war paratyphoid B was the form met with in
Europe, and paratyphoid A in India. Paratyphoid C, which is much less
frequent, is prevalent in the middle East and Mediterranean. With the
admixture of troops, the A and B forms are now equally prevalent. It
has assumed such prominence that inoculation against the A and B para-
typhoid fevers has to be carried out as carefully as against enteric. A
mixed vaccine (T.A.B.) is now usually employed. It contains 1000 million
typhoid bacilli and 500 million of each of the paratyphoid organisms to
the cubic centimetre. A primary dose of 0·5 c.c. followed in 10 days by
one of 1 c.c. is the amount commonly employed for immunisation.
Although various criteria have been cited by different observers as serving
to distinguish the paratyphoids from typhoid fever, the accounts are
extraordinarily contradictory. Except that the former are on the whole
less severe and less protracted there is nothing in the clinical indications
which can be accepted as distinctive. Their differentiation can only be
effected with certainty in the laboratory. But even here considerable
difficulty exists in the case of persons who have been inoculated, though
the quantitative test devised by Dreyer is of some value. The chief
complications are hæmorrhage, perforation, pneumonia and abscess.
Mixed infections of two or more of the varieties of enteric fever are not
uncommon. Treatment is as for enteric.

§ 392. Diphtheria (Synonym : Membranous Croup) is a contagious
fever, characterised by a membranous exudation on the fauces, due to
the Klebs-Loeffler bacillus (Chapter XX). *Symptoms*—The incubation
period is variable, but it is often about two to six days. (1) The onset
is usually gradual (extending over a day or two), but in some cases it is
sudden. The fever is often high, but in others it may not exceed 101
to 103°, and it may even be quite normal (Bristowe) in very mild cases.
The height of the temperature is no guide to the severity of the disease.
The temperature chart does not conform to a regular type, but Fig. 96 repre-
sents a common case. (2) Sore throat is present from the beginning, and
frequently dysphagia except in young children, in whom they are often
absent. On one or both of the tonsils there is a characteristic patch of
creamy white, wash-leather-like membrane situated on an obviously con-
gested surface, and if forcibly removed, this leaves bleeding points. The
patches tend to run together, and may spread on to the pharynx, soft
palate, and uvula. Their occurrence on the soft palate or the uvula is a
diagnostic feature of great value from quinsy. The patient complains
that the neck feels very stiff, and *the glands at the angle of the jaw are swollen.*

This glandular enlargement dates from the recognition of the attack, or even before, and is of importance in the diagnosis. The membrane spreads to the larynx and bronchi in certain cases, and it may also spread upwards to the nose (especially in children). An ichorous discharge from the nostrils in a child lying prostrate and fretful in bed is very characteristic of diphtheria. It may, in rare cases, involve, or start upon the conjunctivæ, genitals, or on the skin at the angles of the mucous orifices. (3) Albuminuria is present in about one-third of the cases (Caiger) before the end of the first week. There may be hyaline casts in the urine which is sometimes suppressed towards the end in fatal cases. (4) Prostration and anæmia are very marked, but the mind usually remains clear to the end,
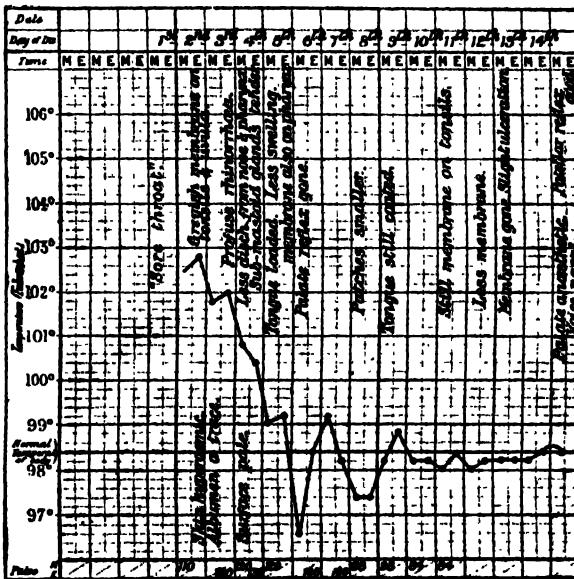
FIG. 96.—DIPHTHERIA.—Male, æt. 9. An ordinary case of faucial diphtheria without implication of larynx. The palate was still anæsthetic one month later. Not followed by paralysis. The different events are indicated on the chart, for which the author is indebted to Dr. F. F. Caiger.

even in lethal attacks. In severe attacks of the disease lassitude and prostration are extreme. Eruptions on the skin are occasionally met with, the commonest being an erythema, or purpuric spots in fatal cases. The Diagnosis of diphtheria may be made by finding the Klebs-Loeffler bacillus in swabbings taken from the seat of the disease. The diagnosis of the sore throat caused by tonsillitis, scarlatina, and diphtheria presents certain difficulties, and is given in the tabular form (§ 130). Follicular tonsillitis is distinguished by the absence of the definite wash-lea patches on the fauces, nasal, or laryngeal passages, and usually the presence of higher fever. There may also be a history of previous attacks, though an inference based on this may be very misleading. Albuminuria, too,

is much less common. *Scarlatina* is distinguished by its abrupt onset, frequently with vomiting, its higher fever, its rash, strawberry tongue, and generally the absence of membrane from the throat. Simple " *croup* " (catarrhal laryngitis) is distinguished by the absence of patches in the throat, but this is often the case in true diphtheria, in which case an appeal must be made to bacteriology. *Membranous croup* is always diphtheritic. *Vincent's Angina* is distinguished by the bacteriological examination (§ 129), and the fact that the patch is usually depressed instead of being raised above the surface.

Etiology.—The disease occurs chiefly in the young, and especially under ten years of age. It is also predisposed to by scarlet fever, measles, whooping cough, and other acute affections. There seems to be a more marked tendency in certain families to contract it than is the case with other infectious maladies. The disease spreads from person to person ; it may be conveyed by instruments, infected handkerchiefs, cups, spoons, slate pencils, and kissing, etc., and it *hangs about a house or district with remarkable tenacity.* Nurses and medical men frequently contract the disease by the patient coughing into their faces. It may also be conveyed by milk ; but there is no evidence that it is conveyed by water. Some hold that the disease is predisposed to by bad air from drains, and undoubtedly, a form of sore throat may be thus developed. It is probable, however, that these conditions only favour the development of the diphtheria bacillus. It is very doubtful if human beings can contract the disease from cats, as has been asserted.

Prognosis.—The case-mortality varied widely in different epidemics, but it used to be on an average from 25 to 50 per cent. Since the introduction of the serum treatment the mortality has fallen to 7 or 8 per cent. in hospital cases (Caiger). The malady is often fatal by the spread of the membrane to the larynx during the first week of the disease in little children. After the first week death may take place by toxæmia, cardiac failure, or other complications. Pharyngeal cases are, in adults, usually mild, and recover in a week or so, but severe cases last two or three weeks. Great care is required even in the mildest cases, lest the membrane should spread, and in the more severe on account of the complications (*q.v.*), particularly cardiac failure. *(*The clinical *varieties,* according to Caiger (*loc. cit.*), are (1) *mild faucial* cases, mostly met with in adults ; (2) *severe faucial* cases, with a tendency to extension, chiefly met with in young children ; (3) " *croup* " or *laryngeal* diphtheria, where the air passages are alone affected ; (4) nasal diphtheria, where the nasal passages only are affected ; (5) diphtheria of *other parts*—cheeks, gums, tongue, lips, conjunctivæ, genitals, wounds, etc.—conditions generally associated with faucial or laryngeal diphtheria.) *Untoward Symptoms.*—The prognosis is unfavourable when the temperature is low in spite of severe local lesions, especially if attended with suppression of urine ; when epistaxis or any form of hæmorrhage occurs, particularly purpuric spots in the skin, such cases being invariably fatal. Speaking generally, the prognosis

will be influenced by the extent, thickness, and persistence of the exudation, the danger being accentuated in proportion to the youth of the patient. Rapid extension of the membrane is also a grave sign, especially when it extends down the larynx, leading to croupy cough, dyspnœa, and cyanosis ; and death takes place in such cases from asphyxia, unless they are promptly relieved (see below), whether by means of antitoxin or by the performance of tracheotomy or intubation. The chief danger in the second week is cardiac dilatation and failure, and the pulse and heart should be closely watched at this time. The appearance of a well marked serum eruption within a week of injection of antitoxin is a favourable sign (J. D. Rolleston). Of the *complications*, certainly the commonest is paralysis, due to peripheral nerve degeneration. It attacks in some degree from 15 to 20 per cent. of the cases (Caiger), and comes on usually about the third or fourth week, sometimes later. The characteristics of diphtheritic paralysis are : (i.) It starts usually in the palate, and therefore nasal voice or dysphagia is the earliest symptom, and fluids taken are returned through the nose. The paralysis is progressive, and tends to involve many of the muscles of the body. Next in order we may get loss of accommodation, squint, loss of patellar reflexes. The most serious paralyses are those of the diaphragm, pharynx and intercostals. (ii.) Motion and sensation are simultaneously affected, though often the sensory symptoms are the first to be observed. The attitude assumed in marked cases is very characteristic—the little patient in protracted cases, if getting up, shambles into the room with drooping shoulders and head bent forward from weakness of the trunk and neck muscles. (iii.) The heart is frequently affected, which sometimes leads to sudden death if the patient be not kept absolutely still. The sounds are weak, and the rhythm rapid and irregular, often of the "cantering" type, in which case the condition is very serious. Vomiting and progressive enlargement of the liver usually accompany such cardiac signs. The temperature is subnormal and the urine suppressed in fatal cases. (iv.) In general terms there is a tendency to complete recovery in a few weeks, though sometimes death occurs—apparently from involvement of the vagus. (2) Broncho-pneumonia, so frequent formerly in laryngeal cases, only attacks about 4 per cent. under modern methods of treatment, but cardiac dilatation, probably due to myocarditis, is a frequent occurrence. (3) Nephritis and dropsy during convalescence are very infrequent, and permanent lesions of the kidney are rare. (4) Otitis media is not uncommon (5·69 per cent. in M.A.B. hospitals 1900–1909). (5) Embolism may occur, secondary to cardiac thrombosis, and give rise to hemiplegia or gangrene of a limb from blocking of the main artery.

Treatment.—The indications are (*a*) to neutralise the toxin in the blood ; (*b*) to inhibit the local process ; and (*c*) to strengthen the constitution to resist the disease. (1) Thanks to the recent advances in science, we now have a powerful *antitoxin* for the control of the disease, and if given early it is capable of completely neutralising the toxin and

arresting the disease. It is a good general rule to give it in all but very slight cases, when under constant medical observation; the antitoxin should be given at the earliest possible moment; doses and methods are given in §§ 418 *et seq.*). (2) For the *local treatment disinfectants are* certainly useful, applied by syringing, or spraying, or swabbing every hour or so, with 1 in 10,000 corrosive sublimate, or carbolic acid (½ per cent.) but the best results have been obtained with chlorine (F. 18), formalin ½ per cent., or sulphurous acid, used by syringing or spraying. Steam inhalations every half-hour and hot applications to the neck give much relief. When the larynx is involved the question of tracheotomy or intubation has to be considered. Statistics used to be very unfavourable, the mortality being 70 or 80 per cent. But in the present day from 70 to 80 per cent. of the cases operated on recover, and either tracheotomy or intubation should be performed promptly whenever the breathing is difficult owing to laryngeal obstruction, provided antitoxin has not been given sufficiently long before to lead one to expect early separation of the membrane. The results are more satisfactory when it is *done early*, and all laryngeal cases should be closely watched for the epigastric retraction during inspiration which indicates severe inspiratory obstruction. It is then desirable to keep the patient in a steam-tent. (3) The *constitutional* treatment consists of stimulating and supporting measures. In all cases the patient should be kept quite still in the recumbent position for fear of the heart failure, which is apt to occur, especially about the tenth to the fourteenth day in severe cases.

Active immunisation by toxin-antitoxin, *i.e.*, a mixture of toxin and antitoxin which is slightly toxic for the guinea-pig, has been largely practised during recent years. It is effected by three weekly doses of 1 c.c. (0·5 c.c. for children under one year), and is said to be protective for some years in most persons. The question of susceptibility to diphtheria is previously settled by the " Schick " test. This consists of the intradermal injection of a minute, though measured, quantity of standardised toxin and noting the occurrence, or not, of a definite local reaction.

§ 393. **Influenza** is an epidemic fever attended by considerable prostration, and usually by catarrh, and a tendency to the development of local inflammations. It has been known for at least five centuries, and has occurred at various times in great epidemics, separated sometimes by many years' interval.

Symptoms.—(1) After an incubation period of one to six days the patient's temperature goes up in the course of a few hours to 102° and 104° F. The onset is frequently attended by severe headache and shivering. The fever generally ends in one to five days with profuse perspiration, and is attended by the pains in the limbs which form such a characteristic feature of influenza. (2) " Catarrh " usually accompanies the fever—*i.e.*, there are redness and watering of the eyes, running at the nose, sore throat, sneezing, and tightness of the chest. (3) Malaise and prostration out of proportion to the amount of pyrexia occur. There is usually a loaded, pasty tongue. (4) Some cases have only the three

symptoms just mentioned, but there is a great tendency to local complications. The *type* of the disease therefore varies according to the physiological system mainly involved. (i.) The *respiratory* tract·is very frequently attacked, and in that case bronchitis and pneumonia complicate the disease. (ii.) The *circulatory* system may be affected by endarteritis, and occasionally, but not often, by other gross lesions. The neurovascular apparatus is, however, specially prone to suffer, causing tachycardia and bradycardia, palpitation, flushings, faintings, perspiration, dyspnœa, and the like. The blood often shows a leucopenia. (iii.) Involvement of the *alimentary* tract may be evidenced by gastro-enteritis, diarrhœa, vomiting, jaundice, etc. (iv.) *Eruptions* on the skin may occur, especially urticaria, erythema, or rose-spots like measles. (v.) The *nervous* system, especially in the aged, is affected for long after the disease, and neurasthenia is particularly apt to supervene. Peripheral neuritis is frequent, and many cases of disseminated sclerosis are attributed to this disease. Depression, prolonged mental dulness, and other symptoms are met with.

The *Diagnosis* is not difficult in typical cases, especially when the disease is prevalent. The short duration of the initial symptoms and the usual absence of rash are sufficiently characteristic. The severe pains in the limbs are very typical.

The *Etiology* is still obscure, though a specific microbe (Pfeiffer's bacillus) has been isolated, which occurs chiefly in the secretion of the respiratory tract. It is uncertain whether this is the only organism capable of causing influenza. Olitzky and Gates have recently isolated a filter-passing organism (Bacterium pneumosintes) from the naso-pharyngeal excretion in the early hours of uncomplicated influenza. One attack confers no immunity from a second. As regards predisposing causes, age has no influence, nor have seasons of the year, nor sanitary conditions. Old and young, rich and poor, all are attacked alike.

Prognosis.—The case-mortality is about 1 per cent. among the old and young together. In middle-aged and elderly people the respiratory type is very apt to end fatally with pneumonia, and undoubtedly many cases presumed to be primary pneumonia are really secondary to influenza. It is fatal only through its complications. The disease itself is usually trivial, and the patient soon recovers. Relapses are not infrequent. The *complications* consist of those mentioned above under types of the disease. The *sequelæ* are prolonged weakness, peripheral neuritis (sometimes attended by tremors), otitis, orchitis, meningitis, and mental derangement.

Treatment.—During the attack, and for a week after the temperature has become normal, the patient should be kept in bed in view of the sequelæ. Sodium salicylate, antipyrin, and quinine will reduce the fever, and relieve the pains in the limbs. A course of vaccine treatment is an advisable prophylactic measure in the case of susceptible individuals. It is well to keep elderly people indoors (or away from infection) during

the season of prevalence of the disease, as they run greater risks from its effects.

§ 394. Rheumatic Fever, Pneumonia, and other Inflammatory Disorders, which usually present well-marked **local manifestations.**—The three fevers just described are those most commonly met with in England, in which the pyrexia may run a continued course, and which have no eruption during the first four days. But it must not be forgotten that certain inflammatory disorders may give rise to pyrexia of a continuous type, and that the usual local signs of these disorders may be absent, at the time when the patient is first seen. It will be well, therefore, to mention those which might be mistaken for an acute specific fever.

(*a*) OBSCURE (so-called) LOCAL [1] INFLAMMATORY DISEASES are mostly met with as complications secondary to fevers. They can usually be detected by a thorough examination of all the organs in the body (§ 376). Nevertheless, certain cases of (1) *pericarditis* or *malignant endocarditis*, or (2) *pneumonia* or *pleurisy*, may be latent—*i.e.*, the usual physical signs may occasionally be wanting or overlooked. (3) Various affections in or around the *throat and nose ;* (5) some *abdominal* disorders, such as peri-hepatitis, inflammation of the mesenteric glands or pancreas, deep-seated abscesses (hepatic, subphrenic, perinephric, tubal), etc. ; (5) certain rare cases of *sarcoma* and *carcinoma ;* or (6) inflammation of the *meninges*, tuberculous or epidemic, may also give rise to an elevation of temperature sometimes unattended by marked local symptoms ; (7) parasitic infections, trichinosis, actinomycosis. Cabot finds that in obscure cases of long-continued fever the causes to be suspected are pulmonary or renal tuberculosis, enteric fever and deep-seated abdominal abscesses, and endocarditis (compare, § 414).

(*b*) Certain obscure GENERAL INFLAMMATORY DISORDERS are attended by pyrexia, which may similarly give rise to difficulties in diagnosis. (1) In *rheumatic fever* and *acute gout* the pyrexia is nearly always continuous. The joint lesions are the cardinal feature in these cases ; but it must not be forgotten that acute rheumatism may commence with inflammation of the pericardium (the structure of which very much resembles that of a joint, and that the joint lesions may not be apparent for several days. (2) There are several conditions special to infancy and childhood which are attended by continued pyrexia : (i.) *Infantile paralysis* (acute anterior poliomyelitis) is attended at its outset by a considerable rise in temperature, which may last for several days or weeks, and be accompanied by restlessness, peevishness, etc. ; (ii.) *rickets* from time to time may have a slight degree of fever, accompanied by a generalised tenderness and profuse perspiration ; and, as just mentioned, (iii.) *meningitis*, tuberculous or epidemic. (3) Examination of the urine may reveal bacteriuria, an unsuspected cause of pyrexia in children. Constipation also may cause fever in both children and adults. (4) *A nervous or hysterical pyrexia* has

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[1] The word " local " is here used in a qualified sense. Many of these diseases with local manifestations are now known to be due to a general infection.

been described, and I have seen the temperature go up in an erratic manner, at odd times, in nervous subjects. But while admitting that the nervous system plays a very important part in the production of fever (as witness the rigors and pyrexia which follow catheterisation), it is difficult to believe that there is not a compound cause in operation in such cases: Only a thorough *post-mortem* and bacteriological examination would enable us to be certain that none of the many obscure foci of inflammation above mentioned were present.

§ 395. **Whooping Cough** (Pertussis) is an acute specific infectious malady, characterised by paroxysmal attacks of coughing, followed by a long noisy inspiration (the whoop). According to Bordet and Gengou the causal organism is a short bacillus which is present in the respiratory mucus. The period of incubation is from three to fourteen days (usually nearer the latter). (1) The onset is marked by a preliminary catarrh, or running from the nose and sometimes the eyes, attended not infrequently by paroxysmal dyspnœa and drowsiness. This premonitory stage lasts from three or four days to a week or more, and may be overlooked. (2) Paroxysms of coughing then set in. Each paroxysm consists of a series of short sharp coughs, followed by a loud *inspiratory* "*crow*," through the narrow chink of the half-closed glottis, and it is often followed by vomiting—a diagnostic feature of value when we have to depend on the mother's account of the case. As the result of the coughing, large quantities of stringy mucus, often blood-stained, are expectorated. After some days the face remains somewhat swollen as the result of the straining cough, and subconjunctival hæmorrhages or epistaxis may also occur. There are no physical signs characteristic of the malady, unless, as some maintain, enlarged bronchial glands can be detected by percussion over the root of the lung. Bronchitic sounds are generally present in greater or less degree. (3) The constitutional symptoms vary considerably in severity. In many cases they are absent, the temperature being hardly elevated, and the child being apparently quite well between the attacks of coughing. In typical cases, however, during the catarrhal stage, there is slight pyrexia. The *Diagnosis* is not difficult, since the paroxysms of coughing are very characteristic, though a typical "whoop" may never be developed. In the absence of the whoop, lymphocytosis aids diagnosis.

Prognosis.—All the symptoms increase for the first ten days, then remain stationary for a few days, and decline during the ensuing two or three weeks. In some cases, however, the attack is much more protracted with a tendency for the whoop to return on taking a fresh cold. It is usually one of the trivial ailments of childhood. It is severe only in very young children, in the weakly and rachitic, or by reason of its complications, of which there are three chief ones—viz., bronchitis, broncho-pneumonia, and convulsions, the two latter being very fatal. Spasm of the glottis may be the cause of sudden death. Otitis media occurs in about 6 per cent. Ulceration of the frenum of the tongue is common, due to the forced protrusion against the teeth in the act of coughing.

Treatment.—In view of the fact, which does not seem to be sufficiently known, that children living near gas-works and bleaching-works do not get the disease, it would be worth while to try inhalations of coal tar. Belladonna is, in my experience, the most useful amongst the drugs, though nothing seems to cut short the malady. It should be given in large doses; children will stand 10 to 20 minims (0·6–1·2) of the tincture if the dose be increased gradually. Antipyrin, carbonate of ammonia, ipecacuanha wine, conium, the bromides, benzyl benzoate and intramuscular injections of ether have also been recommended. Hot baths, bleeding, chloroform inhalations and lumbar puncture may be useful for convulsions. How long a child remains infectious is an important practical question. Infection does not necessarily last as long as the characteristic cough is present, but when, as in some cases, it is hard to

say whether the attacks are typical or not, it is best to take three or four weeks from the commencement of the disease as the duration of the infection. A vaccine prepared from Bordet's bacillus is much used by American physicians. It is said to be useful both for prophylactic and for curative purposes.

§ **396. Mumps (Acute Epidemic Parotitis)** is an acute febrile infectious disorder characterised by inflammatory swelling of one or both parotid glands. The period of incubation is from one to three weeks, and in exceptional cases three or four days longer.

The *Symptoms* are moderate fever (102° F.), subsiding in the course of three or four days to a week, stiffness of the jaw, and difficulty of swallowing, due to swelling and inflammation of the parotid gland. One side is first affected, and is succeeded by the other in about twenty-four hours, or a day or two later. The opening of the duct may sometimes be seen surrounded by a ring of swollen mucous membrane. Sometimes the submaxillary and sublingual glands are also involved. The glands may swell so as to prevent the patient opening his mouth more than a quarter of an inch, and there is sometimes marked salivation. They are acutely tender, and disfigure the patient very much, but the malady is usually a trivial one. The *Diagnosis* of parotitis is very simple. The chief difficulty is between mumps and simple parotitis, such as occurs in enteric and typhus fevers, in abdominal diseases, after laparotomy, or with oral sepsis; but mumps is almost always bilateral, and never suppurates. Care must always be taken to exclude toxic diphtheria, which has often been mistaken for mumps. In epidemics cases are seen of unilateral mumps; and of involvement of the submaxillary glands alone. *Mikulicz' syndrome* is usually mistaken for mumps (§ 9). *Etiology.*—It is almost entirely confined to children and young persons between the ages of five and twenty-five, It is rare in the very young and very old, but is often epidemic and runs through a school. A patient remains infectious as long as there is any definite swelling of the glands. *Prognosis.*—Death from the disease is very rare, and the patient is generally quite well in ten or twelve days at the outside. The chief danger is the swelling of the tonsils and submaxillary glands. In delicate subjects the swelling is slow to disappear. The chief *complications* are (1) orchitis and ovaritis. In these circumstances a very curious phenomenon occurs, for as the testis swells the parotitis subsides. It is the best instance of the phenomenon called "metastasis." The mammary glands may also become swollen and tender. In some epidemics the swelling of the mamma or testicle precedes or accompanies that of the parotid, and in which the former were involved without any parotitis. Occasionally these metastatic inflammations are attended with severe constitutional disturbance, and the affected glands may become permanently atrophied. (2) Pancreatitis, characterised by epigastric pain, nausea, vomiting, and occasionally constipation or diarrhœa; (3) meningitis, usually ill-developed, but sometimes typical; (4) encephalitis; (5) neuritis; (6) otitis interna; (7) œdema of the larynx secondary to submaxillary localisation of mumps; (8) joint symptoms, usually in the form of arthralgia, but sometimes an actual serous or suppurative arthritis occurs. *Treatment.*—The patient should be kept in one room. Warm anodyne fomentations may be applied, and if tension is present, leeches give relief. Diaphoretics and purgatives are useful, and nutrient enemata may be required.

§ **397. Glandular Fever** is an infectious fever occurring in epidemics, in children under fourteen, of cause unknown. It is more prevalent on the Continent than in the British Isles, in France especially. After an incubation period of five to seven days the *symptoms* are : (i.) Sudden onset of fever, 101° to 103° F., with vomiting ; (ii.) transient sore throat ; (iii.) painful enlargement of the lymphatic glands on the second or third day, without redness or œdema of the skin. First the cervical glands are involved, then the axillary, inguinal and mesenteric. (iv.) Abdominal tenderness, with some enlargement of the liver and spleen. The glands begin to decrease in about five days, without suppuration, and the fever may remain till they subside in two to three weeks' time. The blood usually shows a lymphocytosis. *Complications* are otitis media, retropharyngeal abscess, nephritis, and anæmia. The *prognosis* is

favourable. Only four fatal cases have been recorded (P. F. Morse). *Treatment* is symptomatic.

The remaining fevers in this group are PLAGUE, YELLOW FEVER, MALTA FEVER, *which are met with abroad ;* RELAPSING FEVER, *met with in epidemic form only in times of famine ; and* EPIDEMIC CEREBRO-SPINAL MENINGITIS, *which until recent years has for a long time been rare in this country. In* HAY FEVER, DYSENTERY, *and* CHOLERA, *there is some disturbance of the temperature.*

§ 398. **Plague** (Bubonic. Plague, Typhus Bubonicus, Oriental Plague, the Black Death) may be defined as a highly infectious and fatal fever, characterised by inflammatory, glandular, and periglandular swellings, hæmorrhages beneath the skin and from the mucous membranes. The last great epidemic in London was in 1666. Its chief epidemic centres in the present day are Northern India, China, Mongolia, and Uganda. Since 1894 there has been a pandemic over most of the civilised world, and our present knowledge of the disease is therefore greatly increased.

Symptoms.—(1) The incubation period is from two to eight days. (2) There is often a prodromal stage, with depression and pains, but usually the onset is sudden, with shivering, and fever rising to 103° or even 107° F. Mental aberration is not uncommon. The prostration is very marked, and may be accompanied by vertigo, staggering gait, and lethargy, soon passing into the typhoid state. The spleen and liver are usually enlarged. In some cases the speech is halting and staccato, the expression vacant, and the eyes congested; the condition is sometimes mistaken for acute alcoholism. (3) Buboes (inflamed glands) appear in one to five days, usually within twenty-four hours. They may be single, or a group may be affected in one place, femoral or axillary ; sometimes they appear in several parts of the body at once. They may be painless or very painful, and they may suppurate about the seventh day. (4) Petechiæ and subcutaneous hæmorrhages are not uncommon. A distinctive rash is rare, but when present it resembles typhus. There are six principal *varieties*, which prevail in different epidemics : (i.) The *bubonic* variety is the commonest, glandular swellings occurring in quite 70 per cent. of all the cases. *B. pestis* is frequently recovered on blood culture. (ii.) The *septicæmic* type is very fatal : the glands enlarge slightly, but they do not suppurate ; (iii.) an *abortive* form, in which there are buboes without much fever, subsiding in fourteen days ; (iv.) a *fulminant* form, with high fever, little glandular enlargement, vomiting of blood, and death within a few hours ; (v.) a *pneumonic* form, which may be mistaken for bronchitis, influenzal pneumonia, or lobular pneumonia, attended by intense prostration, no glandular enlargement, and death usually on the third to the fifth day, the pulse-respiration ratio being not so much altered as in true pneumonia ; and (vi.) an *ambulant* or mild form, with chronic glandular enlargement, great anæmia, and weakness. The *Diagnosis* is not difficult if sudden onset, marked prostration, mental state, and bubonic swellings be present. The bubo should be punctured before suppuration occurs, when the characteristic bacillus will be found. *B. pestis* is also found in the sputum, practically in pure culture, in the pneumonic form, which variety, indeed, can only be diagnosed by the presence of the bacillus. The sputum in appearance resembles that met with in heart disease. Plague closely resembles typhus in a concentrated form, but a rash is rare in the former, whereas a positive Weil Felix reaction would be obtained before the end of the first week in the latter : and the microbe of plague is distinctive. Inquiry should be made as to the presence of dead rats in the neighbourhood.

Etiology.—Plague is due to the Bacillus pestis, discovered first by Kitasato, and later by Yersin. It was observed that outbreaks of plague were often preceded by a large mortality among rats and other vermin, and it is now known that the bubonic form of the disease is spread by rats. The fleas infesting rats convey the infection to man. The alimentary tract of the flea becomes blocked by a mass of bacilli, the result of growth from infected blood previously imbibed ; some of these bacilli are voided during attempts to suck blood and so pass to a fresh victim, being enabled

to enter through the puncture made by the flea. Filth and overcrowding predispose to plague. The pneumonic form is directly conveyed from man to man by the sputum. Age and sex have little influence.

Prognosis.—The case-mortality in the early periods of epidemics is generally 50 per cent. In well-cared-for white patients the mortality varies from 20 to 40 per cent. In the usual course of bubonic plague death occurs before the sixth day ; or, if the patient is to recover, convalescence starts between the sixth and tenth day. The pneumonic variety is so fatal that of 43,000 cases in Manchuria only three recovered. Prolonged suppuration of the glands may delay convalescence considerably. The course of the disease is very difficult to forecast. Hæmorrhages usually herald death. The *sequelæ* include boils, pneumonia, dropsy, partial paralysis, and mental disorder.

Treatment.—Extermination of rats is part of the prophylactic treatment. The hygienic and therapeutic treatment are as in typhus (see also §§ 416 *et seq.*). The injection of carbolic acid into the glands has been practised with some success, and large doses by the mouth are also recommended. Some advise excision of the glands. Immunisation is now obtained by inoculation of serum if commenced early in the course of the disease. Vaccine injections are also used as a preventive treatment in affected districts.

§ **399. Undulant Fever** (Synonyms : Malta Fever, Mediterranean Fever, Gibraltar Fever) is a disease of subtropical and tropical climates, of wide distribution, with endemic areas, particularly in those countries which border on the Mediterranean, in S. Africa, in the southern portions of the U.S.A., and in the Punjaub. It is caused by a specific organism which is conveyed to man by the milk of infected goats. The goats do not show any sign of ill-health.

Symptoms.—The incubation period is fourteen days, though exceptionally it appears to be very much longer ; the prodromata include malaise, muscular pains, and dyspepsia. A doctor may not be consulted for the first few days, but then the increasing headache, fever, and muscular pains cause the patient to seek advice. The temperature keeps high (102° to 104° F.) for about fourteen days, and may then drop for a few days, only to rise again. After several such undulations the temperature becomes intermittent, with a marked rise at night. The general health of the patient suffers in many ways, the chief symptoms being gastro-intestinal. There are muscular and joint pains, which may be accompanied by considerable swelling, sore throat, sweating, anæmia, enlarged painful spleen, and bronchitis. There are three varieties of the disease. The *malignant* is of acute onset, and runs a rapid course to a fatal termination, preceded by the typhoid state and hyperpyrexia. The *intermittent* variety is of very slow onset, and runs a long course, with elevation of the temperature each evening. The patient does not as a rule make any complaint of specific symptoms until his general health begins to be affected. The *ambulatory* type includes the not infrequent cases in which the micrococcus melitensis is found in the blood of persons who are in no respect ill.

Etiology.—The organisms responsible are the Micrococcus melitensis and the Micrococcus paramelitensis. Goats are affected, and their milk is then infectious.

The *Diagnosis* is arrived at from the clinical signs, by the recovery on culture of the specific organism from the blood or urine, and by the agglutinin reaction of the blood, which should prove positive in a dilution of 1 in 50 in thirty minutes before being accepted as final, adequate controls being essential.

Prognosis.—In the common type the mortality is about 3 per cent. Complications are neuritis, orchitis, diarrhœa, hepatic enlargement, pneumonia, cardiac failure, and hyperpyrexia, the latter being the usual cause of death. The disease may last well into the second year ; the average is 90 days.

Treatment.—Care must be taken to support the heart. Cryogenin gr. x (0·6) is a good antipyretic. Pains in the joints may yield to hot fomentations, but morphia may be necessary. The patient should be removed to a cooler climate if the disease begins in summer. Vaccines, especially autogenous vaccines, give the best results in

expert hands. Prophylactic treatment consists in avoiding goats' milk and cheese. The urine of patients must be disposed of with due antiseptic precautions.

§ 400. Yellow Fever is an acute specific fever peculiar to hot climates and seaport towns, accompanied by jaundice, black-vomit, and the typhoid state.

Symptoms.—(1) The incubation period is short, probably from four to five days. One attack usually renders the patient immune for life. Yellow fever has a sudden onset, the temperature rising on the first day to 101°, 105° F., or even higher, and it remains high for three or four days. At the same time there are severe pains in the head, back and limbs. The pulse does not rise in proportion, and later it becomes distinctly slow. The temperature then falls to normal, or at least remits greatly ; and though it may rise again, the second fever is not so high. The tongue is small, red and jointed. (2) Albuminuria is a constant sign, occurring usually the day after the onset. Granular casts are frequent; The diazo reaction occurs sometimes. (3) In most cases jaundice and vomiting appear about the third day, but in mild cases these may be absent, as may also the yellowness which has given the fever its name. The epigastrium is hypersensitive. In severe cases the jaundice is intense, with petechiæ, the vomit is mixed with bile, and in the later stages with blood, forming the " black-vomit." Hæmorrhages may also occur from the gums, stomach, nose, and bowels. There is no splenic enlargement. There is leucopenia, with moderate increase of the percentage of mononuclears. The liver has a degree of fatty degeneration, and the blood-vessels supplying the stomach and intestines are in a state of degeneration, and readily rupture.

Diagnosis.—Yellow fever has to be diagnosed from many tropical fevers. Important points are albuminuria, slow pulse, epigastric sensitiveness, and slight jaundice. In *malaria* the spleen is enlarged, and the parasite is found in the blood. In *Blackwater fever* there is a bilious vomit, which may cause it to be diagnosed as yellow fever, but it is accompanied by hæmoglobinuria, and no blood corpuscles are found in the urine ; whereas in yellow fever, if the urine is red, it will be found that the condition is due to the presence of blood corpuscles. *Acute Yellow Atrophy* of the liver has a more gradual onset, and is more common in women. Other forms of *jaundice*, including infectious jaundice, must be remembered.

Etiology.—Yellow fever is peculiar to the West Indies, certain parts of America, the Brazilian ports, and the west coast of Africa. It is found only in seaport towns. It rapidly spreads, especially in those parts which are crowded and dirty. It is always worse in the summer months, as a high temperature is necessary for the propagation in the mosquito. A slight frost will destroy it, as at Memphis in 1879. It has been proved that it is transmitted, like malaria, by a mosquito (*Aëdes fasciata*), a domestic species, the causal agent being a spirochæte recently demonstrated and cultured by Noguchi. It is erroneous to state that negroes do not contract the disease. The disease does not spread so rapidly amongst them as among Europeans, probably because many of them have been rendered immune by a previous attack.

Prognosis.—The case-mortality varies in different epidemics from 5 to 94 per cent., and has not improved of recent years. Sometimes the patient recovers uninterruptedly after the fever falls on the fourth day ; in such cases the skin is moist, there is little albumen or vomiting, and little or no yellowness. On the other hand, death may occur with "typhoid state " a few hours after the onset of disease, or from collapse after the fever remits. The prognosis is always grave when the jaundice is intense, the vomiting frequent, and hæmorrhages occur from the stomach or elsewhere.

Treatment.—Prophylactic treatment consists in the destruction of the mosquitoes and their breeding places, and protection from their bites. The provision of piped water supplies has played a great part in stamping out the disease. Patients with yellow fever must be screened from mosquitoes, lest these convey disease to healthy persons. One of the first indications is to diminish the work of the portal system by attention to diet. Vichy water or mutton broth (2 litres of fluid daily) should be given ; no solid food must be taken. Rectal feeding may be necessary. Purgative waters should be given. Symptomatic treatment consists of ice and astringents for the

hæmorrhage, cold sponging and codein for the headache, and strychnine for the heart. No antipyretics should be taken. It is important to induce free elimination by the skin and kidneys. Before the urine diminishes, saline injections should be given subcutaneously, 1000 c.c. daily, in two or three injections. When the temperature has been normal for two days solid food may be taken if the urine is secreting and the gastric irritation has subsided. No work must be undertaken until the pulse resumes its normal rate.

§ 401. **Epidemic Cerebro-Spinal Meningitis** (Synonym : Cerebro-Spinal Fever, Spotted Fever) is characterised by (1) fever, sometimes very irregular at the onset, becoming normal for a day or two, then rising again. It may be remittent, but not often. It is rarely over 102° to 104° F., but may be considerably raised towards the end. The pulse frequency is not always proportional to the degree of fever. (2) Symptoms of irritative intracranial inflammation, such as very severe headache of sudden onset, delirium, vomiting and muscular spasm. Compression symptoms may supervene later. The so-called " Kernig's sign "—*i.e.* when the thigh is flexed at a right angle to the abdomen, the leg cannot be extended because of spasm of the flexors of the thigh—is usually present. (3) There is always retraction of the head, and sometimes opisthotonos may be present, owing to the rigidity of the muscles of the back. Hyperæsthesia, especially along the spine, and severe pain in the back, may be so great that all movement is intolerable. (4) A prominent feature is the presence of some skin affection, very often occurring symmetrically. Herpes labialis or zoster is frequent. On the second day or later a rash of purpuric spots sometimes appears, and may cover the body. Its frequency varies considerably in different epidemics, for in some it has been a rare symptom. Urticaria and erythema may occur. (5) Polymorphonuclear leucocytosis appears early.

Diagnosis.—This disease has to be diagnosed from *tuberculous meningitis*, which has an insidious onset and no eruption. From it and other forms of *meningitis* the best method of diagnosis is by lumbar puncture, when the fluid will be found to be turbid, and to contain the specific diplococcus and polymorphonuclear leucocytes. When an epidemic is present, there is little difficulty in the diagnosis. Care should be taken to exclude *anterior poliomyelitis* with acute onset, in which a stage of cerebral irritation lasting even as long as seven to ten days is not uncommon.

Etiology.—The disease attacks persons under twenty usually, and some epidemics have occurred chiefly among infants, and males more than females. It is most frequent in winter and spring. It is undoubtedly contagious, although much less so than the acute exanthemata. "Carriers" undoubtedly play the chief part in its spread, overcrowding, especially of sleeping quarters, greatly increasing the danger of transmission which occurs as the result of the droplets of secretion being sprayed around during coughing. It is due to a specific micro-organism, the Diplococcus intra-cellularis meningitidis, described by Weichselbaum, which is non-Gram-staining, and best grown on tryp-agar or ascitic fluid. It is found in half the cases in the naso-pharynx, and is carried by healthy subjects.

Prognosis.—The disease has a case-mortality of 30 to 70 per cent. The usual course of the malady is three weeks ; but there are four varieties based upon the duration besides the common form above described ; (i.) the fulminating form, which kills the patient in a few hours or days ; (ii.) the typhoid form, which lasts for several weeks ; (iii.) the form which recovers in a few days ; and (iv.) a chronic form, lasting for months. The prospect of recovery is not good when the disease attacks infants or old people. Amongst the unfavourable signs are the occurrence of hyper-pyrexia, convulsions, irregular breathing, or an unduly prolonged period of illness. The more common complications are inflammation of the joints, optic neuritis, and polyuria. A trace of sugar may appear in the urine. Amongst the sequelæ may be mentioned deafness, iridochoroiditis, panophthalmitis, subacute arthritis, orchitis, chronic hydrocephalus, and transient paralysis of the limbs, aphasia and dementia.

Treatment.—Great success has attended the withdrawal of cerebro-spinal fluid by lumbar puncture, followed by the intra-thecal injection of a reliable anti-serum.

Originally introduced by Flexner in America, this treatment has reduced the mortality from 70 or 80 per cent. to 30 per cent. and lower. It is essential, however, to use the appropriate serum. Mervyn Gordon has isolated four separate serological strains of the meningo-coccus, which occur in varying proportions in different outbreaks. Unless the serum has been prepared from the right infecting strain it is useless. Much of the disappointment in results has been due to this cause, especially in the 1915 prevalence. Lumbar puncture alone, though valuable in itself, is of but limited benefit. About 20 or 30 c.c. of the serum should be given at the earliest possible moment, and repeated daily until the temperature has fallen to normal and the symptoms have abated. Care must be taken that the amount injected is less than that of the fluid removed to lessen the intrathecal pressure. Intraventricular puncture, followed by injection of serum directly into the ventricles, is needed when meningeal adhesions prevent the serum coming in contact with the meningococci. Should the benefit be only partial, a vaccine (preferably autogenous, sensitised if procurable) may then be tried every two or four days. Frequent doses of chloral and bromide, ää 20 grains, may be given at the same time with advantage.

§ 402. **Trench Fever** is characterised by fever of a relapsing type and frequently but by no means invariably, by pains in the shins.

Symptoms.—In the acute type there is high fever for five to eight days, and after an afebrile period there are relapses recurring at five day intervals. In the chronic type the onset shows only a lengthy period of increasing incapacity. In both types the febrile wave is accompanied by severe headache, tenderness of the calf muscles and pains in the shins with nocturnal exacerbations. An erythematous rash in the form of red maculos is frequently found on the chest and abdomen. The spleen is enlarged in about one-third of the cases.

Etiology.—The cause is still unknown, but is probably an intracorpuscular protozoal organism which does not pass through a filter, and is transmitted in the excreta of lice.

Diagnosis.—From Malta and Enteric Fever it is distinguished by the agglutination tests ; from Relapsing Fever by the fact that it does not respond to treatment by the salvarsan group ; from Beri-Beri by the squatting test ; from Influenza by the local signs and the course of the disease.

Prognosis.—Trench fever may run a protracted course, and the patient develop a neurasthenic condition. Disordered action of the heart is a frequent complication.

Treatment.—This is purely symptomatic.

§ 403. **Relapsing** or **Famine Fever** (Synonyms : Recurrent or Relapsing Typhus, Spirillum Fever) is a contagious fever met with in times of famine, ending abruptly on the fifth, sixth, or seventh day, and followed after an interval of one week without fever by a relapse similar to, but shorter than, the first attack. The incubation period varies from five to nine or more days.

Symptoms.—(1) The fever has a sudden onset, with headache, backache, and pains in the limbs. The temperature may reach 108° F., a range which in other diseases is not consistent with life. After remaining elevated for six or seven days, the temperature returns to normal as rapidly as it rose. The fall is preceded and attended by profuse perspiration or diarrhœa, or both. This is followed by an interval of about a week, during which the patient feels exhausted, and the pulse and temperature are subnormal. At the end of this week a relapse occurs which is similar to the first attack, but shorter, lasting three or four days. In rare cases there is a second and even a third relapse. (2) Abdominal pain and tenderness, and great enlargement of the spleen and liver, are present in almost all cases. Jaundice is also very common. Epistaxis is common, and sometimes there is vomiting of blood. Delirium is very rare, but if present is of the noisy kind, and occurs at the crisis. Convalescence is slow. (3) The Spirillum is found in the blood during the pyrexial period, but in the intervals it is only present in the spleen. The *diagnosis* is not difficult, on account of the circumstances under which the disease occurs, and the course of the temperature

Enteric fever, typhus, and *small-pox* cause rash ; *rheumatic fever* is associated with joint lesions. Yellow fever, which it most resembles, produces jaundice, and a diagnosis is only made by the course of the fever and the presence of the Spirillum in the blood in relapsing fever.

Etiology.—Relapsing fever is due to a specific spirillum, which is transmitted from man to man by the body louse or by ticks (in E. Africa and in parts of Persia and Palestine). The disease arises under famine conditions and has been noticed to accompany most epidemics of typhus. One attack does not confer immunity from a second. As regards the *Predisposing Causes,* age has no influence, nor have seasons or occupation.

Prognosis.—The case-mortality rarely exceeds 2·5 per cent. Age has not much influence, but dissipation and debility are unfavourable. Death, which occurs generally at the height of the first attack, is usually due to syncope, from hæmorrhage or from myocardial degeneration. When occurring later, it may be due to complications. Untoward symptoms are : More than one relapse, hæmorrhage, suppression of urine, the typhoid state, cerebral symptoms, or indications of a weak heart. A rapid pulse, a high temperature, and even jaundice, are not unfavourable.

Remedial Treatment.—Salvarsan or one of its substitutes in moderate doses is injected ; if given when the temperature is rising, one dose is usually curative. At the commencement of an attack considerable relief may be given by an emetic or mild purgative. Digitalis may be required for the heart, and paraldehyde for the sleeplessness.

Tick-borne **Relapsing Fever** never assumes the epidemic proportions of the louse-borne disease. It is doubtful whether the relapsing fevers of Algiers, West Africa, India, and America are really specifically distinguishable.

§ 404. Thermic Fever or **Heat Stroke** (Synonyms : Siriasis, Heat Apoplexy, Heat Asphyxia, Sunstroke, Coup de Soleil) is one of the tropical fevers about the pathology of which we know but little. Syncope due to excessive heat is not the same condition.

Symptoms.—The onset is usually sudden, during or after exposure to high temperature with moisture. In some cases there are a few days' prodromata, consisting of headache, malaise, and the passage of an increased quantity of urine. Then a short stage of delirium rapidly sets in, and is immediately followed by coma and high fever (108° to 109° F.). During the stage of delirium the patient is restless, with muscular twitchings and spasms. The stage of coma is marked by a very hot skin, rapid pulse, flushed face, heavy or stertorous breathing, and contracted pupils. In most cases death occurs a few minutes or hours after the onset of insensibility.

Diagnosis.—The coma of *uræmia, diabetes,* and *drugs* (morphia, alcohol, etc.) is known by the absence of high fever. In the coma of *cerebral hæmorrhage* into the pons fever may occasionally be present, but it would not precede the onset of coma. The comatose form of *malaria* is recognised by finding the parasite in the blood, and an enlarged spleen.

Etiology.—All ages and sexes may suffer. It is predisposed to by intemperance, fatigue, and weakness of any kind. It is frequent amongst those who have to perform long marches in the sun of tropical or subtropical climates, and is probably due to accumulation of toxic substances owing to non-radiation of heat from the body.

Prognosis.—The case-mortality is about one in four. Most patients die from failure of respiration after the onset of coma. Favourable cases terminate by crisis, and make a rapid convalescence. Much depends on prompt treatment.

Treatment.—The indication is to reduce the temperature at once, if possible without the use of drugs. Lest malaria be also present, it is best in malarial countries to give an intramuscular or intravenous injection of quinine gr. 7 (0·4) at once, and to repeat it every four hours. The patient must be laid on a stretcher, with a sheet covered with ice placed over him. Iced water should be run over him a few minutes, till the thermometer in the rectum falls to 102° F., or, if much hyperpyrexia be present, to

104° F. Then he should be wrapped in blankets, and stimulants given. Repeat the ice if fever returns. Cold water enemata with 2 per cent. salt and 2 per cent. sod. bicarb. are useful when ice is unobtainable. Avoid strychnine because of the tendency to convulsions.

There are several forms of fever due to Tick-Bites, apart from the relapsing fever above described. The best known of these are :—

§ 405. **Rocky Mountain Fever** (Synonym : Rocky Mountain Spotted Fever).— *Symptoms :* During the incubation period of two to eight days irritation and pain may be experienced in the tick-bites. The fever often commences with a slight rigor, and the temperature rapidly rises to 103°, and later to 105° or even 107° F. ; the maximum is reached by the fifth to the twelfth day. About the third day the eruption appears in the form of macules on the wrists and ankles, which rapidly spread all over the body, including the face, and may become hæmorrhagic. The spleen is palpable and tender, and there may be slight bronchitis and sore throat. Pneumonia is a not uncommon complication. Gangrene of the fingers, etc., may occur. The fever in favourable cases falls by lysis ; if it remains high the patient falls into a typhoid state and does not recover.

Etiology.—This fever only occurs in certain parts of America, and always at a height of at least 3000 feet. It occurs during June and July and chiefly in those whose work or pleasure takes them into the woods and uncultivated regions, where the ticks abound at this time of the year. The tick-bite conveys to the blood an unknown virus.

Diagnosis.—The disease resembles typhoid and typhus. From the former it is differentiated by the eruption, but it cannot always be distinguished from the latter. Exposure to infection by residence in an infected region must be taken into account.

The *prognosis* varies in different localities. In Montana the mortality has been as high as 90 per cent., and in Idaho as low as 2½ per cent. Prophylaxis consists in the avoidance of the places which are tick-infested and by destroying the latter by the application of ammonia, turpentine, etc. The bite may be cauterised with pure phenol.

§ 406. **Kala-Azar** (Leishmaniasis) is a disease found in China, India, Assam, Mesopotamia, and the northern part of Africa, due to the presence in the body of the Leishman-Donovan bodies. An infantile form of the disease occurs throughout the Mediterranean littoral.

Symptoms.—(i.) The disease starts with rigor and vomiting and fever which is usually remittent, but may be intermittent, and lasts for some weeks. Some cases are insidious in onset, being characterised by slight indefinite fever, emaciation and gradual enlargement of liver and spleen. (ii.) The liver and spleen enlarge at the same time. (iii.) After an afebrile period irregular pyrexia appears, of an intermittent type, which lasts on and off during the whole course of the disease. (iv.) Emaciation ensues, which makes even more apparent the enlargement of the abdomen with large liver and spleen. (v.) The patient has an earthy pallor ; bleeding from gums and nose may occur, and, later, œdema and ascites. (vi.) The blood shows little diminution of the red cells, and leucopenia (1000 or 2000 per cubic millimetre), with especial decrease of the polymorphonuclears. The disease is fatal in one to two years in over 90 per cent. of untreated cases ; death occurring usually from intercurrent maladies, especially dysentery.

The *Diagnosis* is made by finding the parasite by liver puncture, or in the later stages, in the blood. As water destroys the parasite the syringe and needle used for the puncture must be dried in alcohol and warmed before use.

Treatment.—Intravenous injections of tartar emetic give excellent results. Two c.c. of a 2 per cent. solution are given twice a week, the dose being increased by 1 c.c. each time till 10 c.c. are given. Continue till the temperature is normal, and no parasites found in the blood obtained by splenic puncture. In children 5 per cent. metallic antimony inunction does good.

Phlebotomus Fever (Synonyms : Pappataci Fever, Three Days' Fever) is a fever

affecting new-comers in the summer months in Herzegovina, Dalmatia, Malta, Crete, Mesopotamia, Egypt, and parts of India.

Symptoms.—After an incubation period of four to seven days the patient has a rigor, followed by severe headache, fever, and severe pain in the eyeballs and brow, back, and calves of the legs. The eyes are congested, the face flushed, the tongue foul. The fever lasts from one to five days, most often seventy-two hours. The disease is seldom or never fatal, and one attack confers immunity.

Etiology.—The poison reaches the blood of man by the bite of a sand-fly, the phlebotomus papatasii, which abounds in the summer in the above-mentioned districts. There is evidence that the parasite is a spirochæte which in one stage of its existence can pass through a Pasteur filter.

Treatment.—Opium given early is the best drug. For prophylactic treatment apply eucalyptus or cassia oil to the exposed parts, and fumigate the rooms with sulphur. The insect bites at night, and is so small that it can penetrate the meshes of a mosquito net. It dislikes sunlight and wind.

Rat-Bite Fever has long been described in Japan as occurring after the bites of rats and cats, and was known by the history of a bite, a purple-coloured eruption, enlargement of the regional lymphatic glands, and fever of a relapsing character. Cases have also been met with in Europe and America. (i.) There is a history of a rat-bite which heals slowly ; (ii.) four or five weeks after there is pain and swelling at the situation of the bite, with fever, which may reach 105° F. ; (iii.) the fever recurs at intervals of days, weeks, or months. It may last only a day, or a week, assuming an intermittent type ; (iv.) the fever in some cases is accompanied by an erythematous eruption ; the blood shows a moderate leucocytosis. The disease is nearly certainly due to a spirochæte, and reacts readily to salvarsan and similar preparations.

Japanese River Fever (Synonym : Tsutsugamushi Disease).—The *symptoms* resemble those of Rocky Mountain Fever. On the first or second day there is a tender, non-suppurating enlargement of the lymphatic glands, and near some groups there are found one or more small black scabs surrounded by an inflammatory redness. On the sixth or seventh day a papular eruption appears first on the face ; it spreads downwards, and lasts four to seven days. During the second week of the fever the scab falls, leaving a punched-out ulcer, which may take some weeks to heal. Conjunctivitis is present early in the disease.

Etiology.—The disease occurs only in certain flooded valleys on the west coast of the largest island in Japan and in Formosa. It may be transported by corn, hemp, and other articles. It is due to the bite of a mite which bores into the skin. A Rickettsia-like body is thought to be the causal organism.

The *Diagnosis* is made from a consideration of the locality and by the skin lesions. Otherwise it closely resembles typhus and Rocky Mountain fever.

The *Prognosis* is good in the young, and in second and third attacks. The mortality is 30 per cent. *Treatment* is symptomatic.

Psittacosis is a disease epidemic among parrots, due to a bacillus of the coli group. It is conveyed to man by handling the birds, and is seldom communicated from man to man.

Symptoms.—The incubation period is seven to twelve days. The onset is acute or gradual, more usually the latter. The temperature rises to 102° to 104° F., and the spleen is enlarged. Rose-coloured spots appear on the skin, and the patient falls into a lethargic semiconscious condition, in which he may remain several days. The disease may terminate in recovery after two or three weeks. An atypical pneumonia is a common and fatal complication ; the death-rate is 35 per cent.

The *Diagnosis* is made from the presence of sick parrots in the house of a patient affected with an obscure fever and pneumonia. The bacillus may be recovered from the sputum.

Treatment is symptomatic. Prophylaxis consists in the quarantining of all parrots

imported from South America and the destruction of all found to be infected, as well as of their cages, etc.

Hay Fever (Hay Asthma), especially the constitutional variety, Dysentery, and Cholera, give rise to a certain amount of pyrexia of a continued type.

HAY FEVER (§ 153) is recognised by the violent attacks of sneezing.

DYSENTERY (§ 244).—Acute dysenteries are sometimes attended at the onset by some degree of pyrexia, but much the most important symptom is diarrhœa.

In CHOLERA (§ 245) the abdominal cramps, collapse, and diarrhœa are the leading symptoms. During the collapse stage the temperature may be as high as 105° F. in the rectum, although in the axilla and mouth it is subnormal. In the reaction stage, if the patient lives, there is usually a degree or so of pyrexia lasting from a week to a fortnight.

Finally, there are several diseases which in their typical forms belong to Group III, or, belonging to Group I, are seen perhaps before or after the eruption comes out, which may present pyrexia of a continued type. It is well in all cases of difficulty or doubt to remember this, and to pass in review the members of all three groups.

GROUP III. INTERMITTENT PYREXIA

§ 407. In this group of diseases the pyrexia is of an INTERMITTENT (or remittent) type—i.e., the temperature drops at regular or irregular intervals to normal (or nearly to normal). This group is distinguished from Group I by the complete absence of eruption. It is distinguished from Group II mainly by the wide variations of the temperature.

Common.		Rare.		
I. Malaria	§ 408	Malignant endocarditis ..	..	§ 46
II. Latent tuberculosis ..	§ 410	Lymphadenoma	..	§ 438
III. Visceral syphilis	§ 412	Pernicious anæmia ..	..	§ 433
IV. Acute septicæmia	§ 413	Leukæmia	..	§ 437
V. Subacute septic conditions	§ 414	Opium habit ..	..	§ 560
VI. Enteric and paratyphoid		Trypanosomiasis	..	§ 415
fever (some cases) and		Trichinosis	..	§ 475
occasionally influenza ..				

The clinical investigation of these diseases is often attended by considerable difficulty. Malaria, which may be regarded as the type of this group, is essentially a *paroxysmal pyrexia*, each paroxysm having three stages (cold, hot, and sweating), and each paroxysm being typically *separated by one or more days' interval of health*. TUBERCULOSIS and SYPHILIS have a daily rise and fall, and are good examples of *regular diurnally* intermitting pyrexia. ACUTE SEPTICÆMIA, on the other hand, is noted for the *irregular* character and wide range of its temperature and the severity of the rigors. CHRONIC SEPTIC CONDITIONS occupy a position midway between these two types—regular and irregular intermitting pyrexia. In a given case of intermitting pyrexia which has arisen in a tropical or subtropical climate, malaria, undulant fever, or tropical liver abscess are probable, but in England the commonest cause is probably latent tubercle. Tubercle as a cause of this type of fever is nearly as common in the tropics as elsewhere. The SERUM REACTIONS aid us to some extent in the diagnosis of this group.

Turning to the rarer diseases, which must always be kept in mind, MALIGNANT ENDOCARDITIS is chiefly remarkable for the *long course* it may run. In LYMPHADENOMA we find the enlarged *glands*; and in PERNICIOUS ANÆMIA the patient is more often a male, the skin is very sallow, and the blood picture is characteristic.

It follows therefore that if we have a patient's temperature chart before us, and it shows definite intermissions or remissions, the disease will belong to one of three sub-groups :

A. REGULAR INTERMITTENT PYREXIA, with one or two days' INTERVAL, which contains only one disease—Malaria ... ... § 408

B. REGULAR INTERMITTENT PYREXIA occurring DAILY, such as Tuberculosis, and Visceral Syphilis ... ... §§ 410 *et seq.*

C. IRREGULAR INTERMITTENT PYREXIA, such as Septicæmia, and other pyogenic processes ... ... §§ 413 *et seq.*

§ 408. **Malaria** (Synonyms : Ague, Intermittent Fever, Remittent Fever, Jungle Fever).—Malaria is a non-contagious fever, occurring in paroxysms with complete intermissions, due to the malarial parasite introduced by the bite of a mosquito.

Symptoms.—As a rule the fever comes on suddenly without warning. The period of incubation varies considerably, but is most often ten days ; while in some cases there may be an interval of months or years after exposure before the disease develops. A typical paroxysm has three characteristic stages. First there is a *cold stage*, in which the patient shivers or has a rigor, and feels cold, though the temperature is steadily rising ; the skin looks cold, sometimes livid, and the nails are blue. It lasts from one-quarter to two hours, or so, and is followed by the *hot stage*, in which the temperature goes up to 103° to 106° F. It begins with flushing of the face, and is attended by headache, pains in the back and elsewhere. It lasts three or four hours, and is followed by the *sweating stage*, in which the perspiration is so profuse that the bed-linen may be soaked. This stage lasts one or two hours, and is accompanied by a fall of temperature. The spleen enlarges during the attack. The stages may be shorter or longer. The sweating stage is followed by an interval, during which the temperature is normal, or subnormal, and the patient is fairly well, except for great lassitude and indigestion. In the commonest type of the disease (Tertian Ague, Fig. 97), there is an interval of about twenty-four hours between the paroxysms, which, if untreated, may recur for weeks.

Varieties of Malaria.—Malaria fever may vary in two ways : (*a*) According to the duration of the interval between the attacks, or (*b*) according to the intensity ; both of which probably depend on the species of the plasmodium. (*a*) There are three types of periodicity (Fig. 97) : (i.) *Quotidian* fever, in which the paroxysm occurs daily, is due to a double infection of tertian or quartan fever ; (ii.) *Tertian* fever, in which attacks occur every other day ; and (iii.) *Quartan* fever, in which the attacks occur with two days' interval. Various compounds of these occur. (*b*) *Pernicious* malaria is the term given to the severe forms, the chief varieties of which are (i.) the *comatose*, in which the patient suddenly passes into coma ; (ii.) the *hyperpyrexial*, in which the temperature rises suddenly to 107° or 112° F., and death occurs in a few hours ; (iii.) the *algid*, which

resembles the algid stage of cholera; (iv.) Remittent Fever; and (v.) Blackwater Fever. The *œstivo-autumnal* parasite is present in the pernicious forms of malaria.

Diagnosis.—Malaria is rarely mistaken for other diseases; but the other disorders attended by intermitting pyrexia about to be described are very frequently mistaken for malaria. *Clinically*, this mistake would be avoided if it were remembered that tertian or quartan periodicity is absolutely pathognomonic; it occurs in no other disease. In leprosy and all the diseases mentioned below the intermission is daily. *Therapeutically*, the diagnosis may be established by full doses of quinine; if this be given intramuscularly, and fail to relieve, the attacks are certainly not malarial.
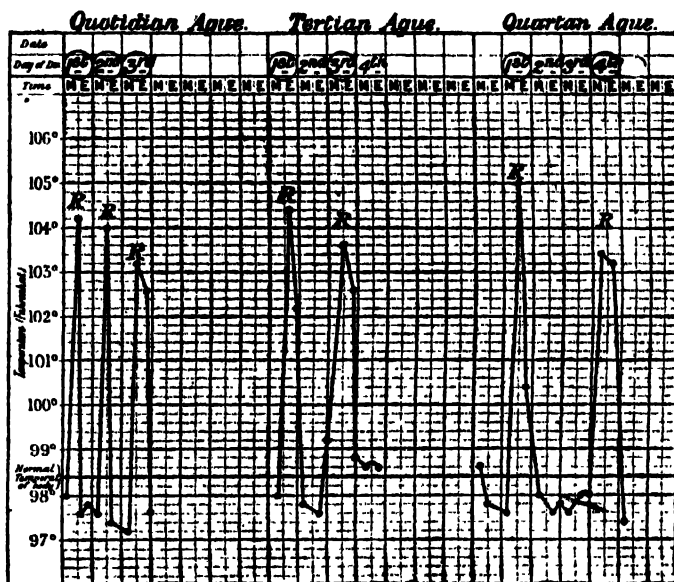
FIG. 97.—TYPES OF AGUE.—Quotidian (daily); Tertian (every other day); and Quartan (every third day). "R" indicates the rigor which ushers in the cold stage.

The *microscopic* recognition of the parasite in the blood requires considerable experience, but it is always possible to find it in blood-films, provided the patient has not taken quinine for several days. If quinine has been given, a tentative diagnosis may be made if the blood shows leucopenia, diminished hæmoglobin and red cells, and increase of large mononuclears. Enteric fever and many other conditions belonging to Group II, when occurring in a malarial subject, are apt to assume a malarial or intermitting type of pyrexia.

Etiology.—Age and sex have no real influence. The disease is most prevalent at the latter part of the rainy season. One attack predisposes to a second one; indeed, when once a person has contracted malaria, he is always liable to it for many years. The *exciting* cause of malaria is a

parasite—the plasmodium (see § 429). It is introduced into the blood of the patient by the bite of a mosquito (the females of several species of *Anopheles*), which serves as the true host for the parasite. The pioneer work of Laveran, Manson, Ross and Nuttall identified the causal agent and proved the method of its transmission by the bite of the mosquito. There are three forms of human parasite known—(*a*) the tertian ; (*b*) the quartan ; and (*c*) the malignant tertian. The type of the fever depends upon the time required for the sporulation of the parasite, since the onset of the pyrexia corresponds to the day on which sporulation is completed. Thus the parasites of tertian and malignant tertian ague complete spore formation in two days, that of quartan ague in three. The disease is endemic in certain districts, which are called malarial, and these districts are generally situated in tracts of country which are marshy, or where the soil is moist and covered with pools of water and decomposing vegetable matter. It is still to be met with in Italy, Greece, Russia, and North America, and in some parts of England—*e.g.*, Kent—but it is chiefly in the uncultivated tracts of India, Assam, Africa, Gallipoli, Asia, Central and South America that the disease is prevalent. It disappears from a district when the soil is drained and cultivated. Standing water, especially in puddles, seems to be a necessary condition, together with a moderately high temperature for the development of the mosquito and the propagation of the parasite within it. Persons newly arrived in a district are especially prone to contract the disease.

FIG. 98.—MOSQUITOES settling on a wall. There are two chief types of mosquitoes—Anopheles and Culex—easily differentiated by their attitudes when resting upon a wall. Anopheles is the more dangerous one, and is recognised by its spotted wings and its tilted attitude. Its larvæ lie flat on the surface of puddles, whereas Culex larvæ lie more perpendicularly, and if disturbed rush to the bottom of the pool. Anopheles larvæ are found in puddles which contain algæ and which are too large to be dried up in a week (time needed for the mature insect to be hatched). They are not found in pools which contain minnows, nor in shallow rain pools that are easily dried up. In certain districts they may be found even in rapid streams. Kerosene oil (about 3l to a pool of 1 square yard) killed all larvæ in six hours.

Prognosis.—Death usually occurs from complications, without which malaria is not a very fatal disease. The most favourable type of case is that in which the pyrexia runs a typically intermittent course. The gravest, and happily the rarest, is that form in which the pyrexia is continued or only remittent. Coma or delirium, hæmorrhage from the stomach or bowels, and choleraic diarrhœa with cramps are unfavourable complications, and if collapse sets in after the hot stage a fatal termination

is usual. (i.) Great weakness and anæmia are common results of the disease, and in time, especially if untreated, the patient develops the typical cachexia of malaria. Pigmentation of the skin is a marked characteristic of this cachexia ; the distribution of pigment is general, but is especially evident around the eyes. On one occasion when I joined a passenger ship from the East this periorbital pigmentation was so marked in one of the passengers that I thought he must have been fighting, until I learned that he had been a victim of malaria. This is doubtless accounted for by the deposit of blood-pigment granules which are so constantly found in the blood. (ii.) Enlarged spleen—" ague-cake " (§ 288, V)—is a usual sequence, and rupture of the organ occasionally takes place. (iii.) Jaundice, due usually to hepatitis, is one of the more serious complications, and the liver after many attacks becomes enlarged. Hæmatogenous jaundice, due to the destruction of red cells, may also follow fever of long duration. (iv.) Pulmonary complications, pleurisy, bronchitis, and pneumonia are common, particularly in those who have returned to live in cold climates.

Treatment.—Quinine is a specific for this disease. As far as possible, it should be given by the mouth, and when the attack is not of a serious nature, its administration should be delayed till the temperature falls, and meanwhile the patient made as comfortable as possible. Morphia by injection is justifiable at times. For oral administration quinine should be given in a powder, or freshly dissolved in a mixture, as pills and tabloids are useless if hard and insoluble. The bisulphate and bi-hydrochloride of quinine and euquinine are good preparations, gr. 5 to 10 (0·3 0·6) thrice daily after meals. During the acute stage of the disease as many as 20 to 30 grains (1·2–2) daily may be given. A mercurial aperient should be administered at the onset. In the mild tertian and quartan fevers, the drug should be given four hours before the attack—*i.e.*, before sporulation. When prompt action is required (as in the pernicious form) the quinine should be administered intramuscularly or intravenously ; in the pernicious form 15 grains (1) of quinine are given. Intramuscular injections are also used when there is much gastric disturbance. Sterilettes are procurable containing the correct dose of the drug dissolved and ready for use for intramuscular or intravenous injection. In algid conditions, hypodermic saline injections are called for. Warburg's tincture, which contains a small proportion of opium, has been found to act better than quinine in certain cases. The drug should be continued in small doses for some time after the subsidence of the fever. For the resulting anæmia, iron and arsenic are necessary ; sulfarsenol is admirably adapted for this purpose. For the " ague-cake " red iodide of mercury ointment should be rubbed in over the enlarged spleen. The indications for prophylactic treatment are based upon the etiology. In order to get rid of the breeding-grounds of the mosquito, marshy tracts, swamps, and roads must be drained ; cisterns and wells must be screened, and kerosene oil should be poured upon all stagnant pools. High sites should be selected for

sleeping. Oil of cassia and citron should be rubbed on exposed parts. Mosquito nets are essential, and wire netting for rooms and houses can be obtained. Quinine should be taken (5 grains (0·3) daily) as a matter of routine by those who live in the mosquito-infected districts.

§ 409. **" Blackwater Fever "** (Synonym : Hæmoglobinuric Fever), so named from the colour of the urine, is almost certainly the direct result of malarial infection, and occurs only in countries where remittent fever is general.

Symptoms.—In a typical attack the onset is marked by·rigors, and the temperature ranges from 103° to 105° F. The urine which is passed is of a dark red colour, turning to black, due to the presence of hæmoglobin ; it is scanty in amount and of high specific gravity. There is bilious vomiting, which may be extremely severe, and accompanied by intense jaundice. • As the fever falls, the urine clears ; then a new paroxysm of fever may set in, with a return of the hæmoglobinuria. The liver and spleen may be enlarged. During the paroxysm there is great destruction of red blood corpuscles, and the blood shows poikilocytosis. *Etiology.*—This fever is endemic in certain areas of Africa, America, Asia, the Balkan Peninsula. What determines the onset of the paroxysm is not known. Some have thought that the hæmoglobinuria is related to the administration of quinine, but only in patients who have suffered previously from malaria.

Diagnosis.—This disease may be mistaken for yellow fever. Malaria parasites are rarely found during, but can be found before or after, an attack.

Prognosis.—The case-mortality is 25 per cent. Frequent relapses are certain to occur if the patient remains in the endemic district, unless he succeeds in protecting himself from recurrence of malaria. Even if he return home, he is liable to have attacks of hæmoglobinuria, though these may be accompanied by little or no fever. In severe cases there may be profound prostration, with all the symptoms which accompany a profuse hœmorrhage. Death may result in this way, or from syncope, or collapse. There may be suppression of urine, and death with symptoms of uræmia.

Treatment.—Except during the attack, quinine should be given only with great caution, beginning with gr. i. (0·06) t.i.d. Saline solution must be given *per rectum*, intramuscularly, and if necessary intravenously. The patient must be kept at rest, and water must be freely given. Adrenalin by mouth will sometimes control the vomiting.

§ 410. **Latent Tuberculosis.**—Tuberculosis is said to be latent when the usual physical signs or local manifestations are wanting. In all cases of unexplained intermitting pyrexia in this country, one of the first things to be suspected is tuberculosis in some parts of the body. It may be very deeply seated, but it is a useful clinical axiom to remember that no *active tuberculosis can exist in any part of the body without the occurrence of a daily intermitting pyrexia.* Moreover, the degree of the fever is a fair indication of the activity of the process. The chart is a typical one ; the temperature drops each morning to (about) normal, and rises each evening one, two, or more degrees, occasionally *vice versa.* The physical signs may be altogether wanting, and the patient, perhaps, only seeks advice on account of the weakness, dyspepsia, and other vague symptoms. Such a condition may go on for weeks without any local manifestations, as in the cases referred to under Tuberculous Meningitis. The lungs, kidneys, peritoneum, and various organs may be affected. (1) The commonest locality in adult life is the *lungs.* In this case physical signs usually appear which resemble bronchitis or simple pulmonary congestion, for

which diseases it is apt to be mistaken (§ 96). (2) The *meninges, peritoneum*, and other *serous membranes*, are perhaps the commonest positions in childhood in which tubercle may be deposited without definite signs. (3) In the *kidney*, tuberculous pyelitis may be readily ov.rlookcd, and in suspicious cases the urine should be carefully examined for traces of pus and tubercle bacilli (§ 331). (4) Tubercle may also be latent in other situations, such as the cranium, spine, intestines, and other viscera; and, finally, the tuberculous process may be generalised, and give rise to the condition known as *Acute General Tuberculosis*. In the diagnosis of tuberculosis we may seek the aid of certain blood reactions (see § 110),

§ 411. **Acute General Tuberculosis** [Synonyms: Acute Miliary Tuberculosis Typhoid Tuberculosis) may be of the meningeal type, usually known as tuberculous
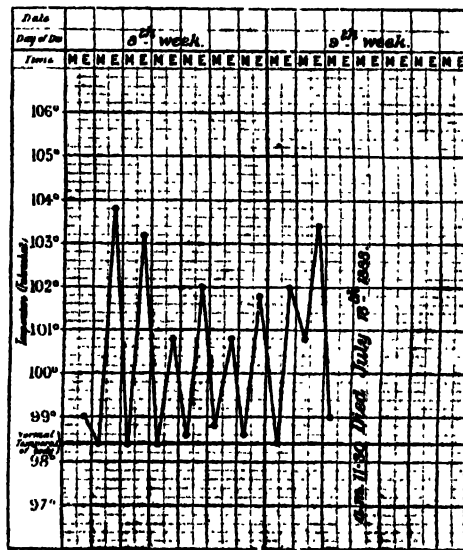
FIG 99.— ACUTE MILIARY TUBERCULOSIS.—Geo. W——, æt. forty-nine, admitted to the Paddington Infirmary, July 9, and died July 18, 1888. Seven weeks' history of vague illness before admission, during which time there was profuse hæmoptysis on one occasion. The signs in the chest were very indefinite during life. After death the lungs were sparsely studded with miliary tubercles. The liver and peritoneum were also dotted with tiny tubercles, hardly visible to the naked eye.

meningitis, and described under that title; of the pulmonary type (*vide* § 96); or of the typhoid type, with which we are now concerned. It is characterised by intermitting pyrexia, prostration, and a tendency to the typhoid state—due to a generalised infection of the body by the tubercle bacilli.

Symptoms.—(1) The onset is insidious. The patient complains perhaps of nothing but lassitude, which is attended by feverishness of a typical intermitting type, and perhaps bronchial catarrh. The temperature each morning may be normal, that in the evening raised one or more degrees. The inverse type—*i.e.*, a lower temperature in the evening than the morning—is said by some to be more frequent in this than in other forms of tuberculosis.[1] In very rare cases the highest daily temperature does not

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[1] According to Reinhold (quoted by Osler, *loc. cit.*), 18 per cent. of tuberculosis cases present an inverse temperature.

rise above normal. The patient complains of lassitude, which gradually increases, and in the course of a few weeks he has wandering, muttering delirium, at first only at night. Maniacal delirium is rare. The typhoid state supervenes towards the end. (2) The respiration is always increased in frequency. The pulmonary signs, which are generally present, have been mentioned in § 96. (3) As a rule there are no marked local manifestations, but, according to the chief seat of mischief, various other signs may be elicited, such as paralysis of the cranial nerves, peritonitis, pleurisy. The spleen is nearly always enlarged.

Diagnosis.—(1) The presence of bacilli in the sputum is pathognomonic, and these should be looked for repeatedly in all cases of " *bronchitis* " attended by an intermitting pyrexia, especially in young adults. Most cases of acute miliary tuberculosis in the early stage are admitted to hospital as bronchitis, in the later stages as enteric fever. (2) The course of the disease may bear so close a resemblance to *enteric fever* that Niemeyer (before the discovery of the tubercle bacillus) stated that these disorders might be indistinguishable until the patient reached the dead-house. The Ehrlich-Diazo reaction occurs in both enteric and acute tuberculosis, but not the Widal reaction. Choroidal tubercles are sometimes visible on ophthalmoscopic examination, and if present settle the diagnosis.

Etiology.—The disease is due to a general dissemination of the tubercle bacilli throughout the body. These may have been introduced from outside, but far more frequently can be traced to some chronic or subacute focus in the patient himself, such as an old caseous or fibroid gland which appeared to be dead, or an old quiescent spot in the lungs or elsewhere.

Prognosis.—The disease is uniformly fatal in the course of four to eight or more weeks. Death occurs by coma, sometimes by pulmonary or other complications. The height and range of the temperature is a fair measure of the virulence and activity of the morbid process.

Treatment.—In such widespread mischief no treatment is of any avail. As regards prevention, it should always be remembered that convalescence from pulmonary tuberculosis should be very thoroughly re-established before treatment is stopped.

§ 412. Visceral Syphilis.—It is now generally recognised that syphilis is a specific contagious disease like small-pox. There are two different stages of syphilis at which intermitting pyrexia may occur. (*a*) At the first development of the primary roseolous eruption there may be some elevation of temperature.[1] This is generally overlooked, but at other times it may be accompanied by thirst, loss of appetite, and shivering. It always occurs within sixty-five days of the date of the infection, and is only present if no mercury be given. (*b*) In the later secondary and tertiary stages of the disease intermitting pyrexia may occur in connection with syphilitic periostitis, or gummata of the internal organs.[2] This is a not infrequent occurrence in the course of clinical work, and

[1] This has only been generally known of late years, but it was first pointed out by Guntz in 1865, and called " general syphilitic fever." Lancereaux also pointed it out in 1866, and stated that it much resembled quotidian ague. He referred to several cases.

[2] Two cases are reported by Dr. Alfred Dufferin in the Clin. Soc. Trans., 1869. The following year a Committee reported nine cases. Dr. Bristowe reports a case of gumma of the liver in a lad of sixteen, in whom the temperature went up every evening 2° or 3°, the cause being overlooked until interstitial keratitis was discovered, and iodide was given (Clin. Soc. Trans., vol. xix.). The author has records of six similar cases in which the leading symptoms were intermitting pyrexia, anæmia, and signs referable to the liver or spleen, all of which rapidly disappeared under iodide (see also *Clin. Journ.*, December 1, 1897, p. 87).

syphilitic lesions of this kind are always to be suspected in cases of prolonged intermitting pyrexia, especially if it be attended by anæmia. The morning temperature is normal, but in the evening it goes up one, two, or more degrees (Fig. 100). There may also be rigors, nocturnal sweating, and paroxysms of pain in the joints, unrelieved until iodides are given; then the symptoms speedily subside. In obscure cases careful investigation should be made of the eyes, liver, ribs, clavicles, and other bones, and iodide of potassium tried. In rare instances the fever may be continued and simulate typhoid.
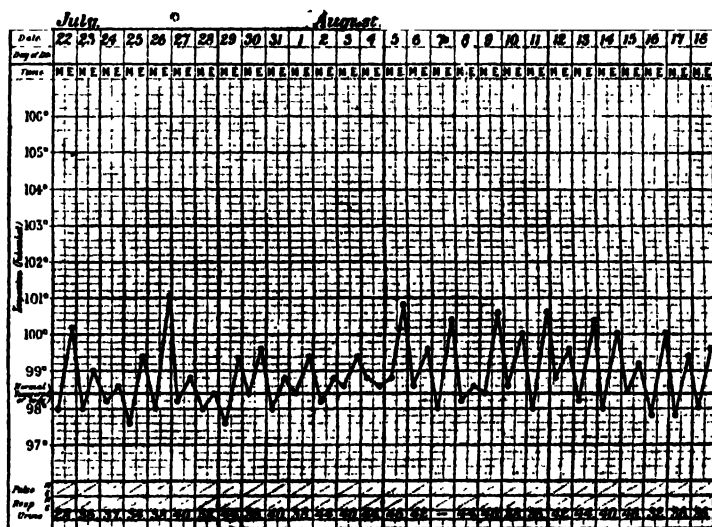
Fig. 100.—VISCERAL SYPHILIS.—Annie L——, æt. sixty-six, admitted to the Paddington Infirmary, July 22, 1889 (?). The temperature subsided under iodide in large doses, but she ultimately died of exhaustion and hypostatic pneumonia. P.M.—Gummata of liver and bones, hypertrophic cirrhosis, widespread fibrosis of organs.

§ 413. Acute Pyæmia, or Septicæmia,[1] is a disease characterised by a wide range of temperature, accompanied by rigors and sweating, due to the direct infection of the blood—usually through some breach of surface in skin or mucous membrane—by a pyogenic micro-organism.

The *Symptoms* are (1) pyrexia, which runs a very characteristic course, and is distinguished from all other diseases not of septic origin by the *wide* and *very irregular* range of the temperature (Fig. 101). The remissions may occur several times a day, and have not the diurnal regularity which marks the two preceding classes of disease (§§ 410 and 412). There may be as much as 6° or 7° difference between the temperature in the course of a few hours. When at its highest point, the temperature is accompanied by rigor, followed by very profuse perspiration and a rapid

[1] There is still some confusion in the use of these terms, but for clinical purposes they may be regarded as synonymous. In former times, when localised deposits of pus occurred, the former term was generally applied; when these were absent, the latter.

fall. The pulse is rapid and compressible, and the prostration and lassitude are very marked. The mind is clear at first, and remains so for a considerable time, but towards the end there is a tendency to the typhoid state. (2) Nausea, vomiting, and diarrhœa are common, the skin is sallow, and there is often jaundice. (3) Later on in the disease emboli may occur in different parts of the body, especially in the lungs, where they give rise to a generalised congestion and patches of pneumonic consolidation or gangrene (as in the case given in Fig. 101), and in the liver and spleen, and deposits of pus may occur in or around the joints or in other parts of the body. The serous cavities may also contain pus, constituting empyema or pyo-pericarditis. The occurrence of albumosuria is an indication of a focus of pus in the body, and this may be an aid to diagnosis; so also are the leucocytosis and other changes in the blood (Chapter XVI).

Acute Osteomyelitis (or, as it used to be called, Acute Periostitis or Acute Necrosis) is a pyæmic process which may set in very suddenly, usually after an injury to one of the superficial bones, generally the tibia. In children there may be no history of injury. The diagnosis is easy when the tissues round the diseased bone are swollen, but during the first day or two of the disease pain is often complained of near a joint, and may lead one to diagnose rheumatic fever.

The *Diagnosis* of septicæmia is easy when there is an external wound or abrasion, and should never be difficult on account of the wide variation of the temperature, coupled with the rigors and the sweats. The chart of a typical acute case is like nothing else. When due to some internal cause, it may resemble malignant endocarditis, enteric fever, pneumonia, ague, remittent fever, and acute rheumatism. But when carefully recorded temperatures of several days are available, and a thorough examination of the organs is made, the diagnosis should not be difficult. Blood cultures reveal the nature of the invading organism, usually a staphylococcus or a variety of streptococcus.

Etiology.- -A cause--*external* or *internal*- should be carefully sought for. Among *external* sources, unhealthy wounds were, before the introduction of Listerism, a prolific source of this disease, and the patients in the surgical wards were decimated by it. A mere scratch is sometimes sufficient for the introduction of the micro-organisms, and sometimes the most trivial operations are followed by pyæmia. The source of infection may arise from some *internal* condition. The internal sources are very numerous—sometimes it is caries, especially of the mastoid bone, sometimes periostitis, or osteomyelitis, sometimes an ulcer and other breach of surface in the mucous membranes. Ulceration of the biliary passages and of the urinary passages are frequent sources of infection. Special attention should be directed to the vermiform appendix (see Appendicitis, § 199) and the uterus. *Recent abortion, perhaps criminally procured, should always be borne in mind when a young woman is admitted with septicæmia.* After recent parturition, the uterus resembles an open wound, and offers a large surface for the absorption of the pyogenic

organisms; hence the frequency with which septicæmia complicates par-
turition unless the most scrupulous cleanliness has been observed. The
disease is then called Puerperal Fever, or PUERPERAL SEPTICÆMIA.* When
the poison is derived from a previous case of puerperal septicæmia it is
specially virulent and fatal. Among the *predisposing causes* overcrowding,
bad ventilation, want of cleanliness, and other unhygienic and septic
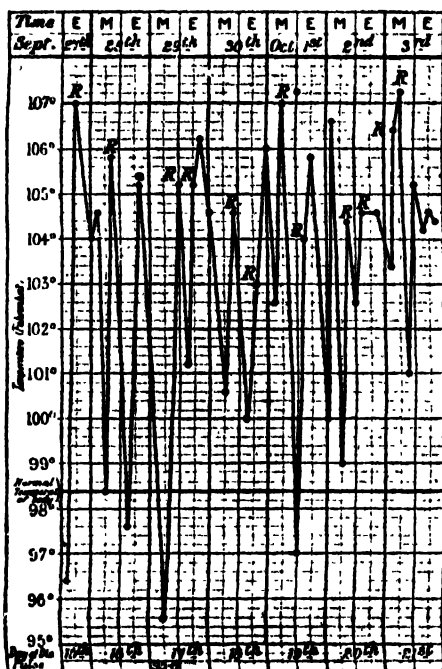conditions, are among the most fruitful.

FIG. 101.—ACUTE SEPTICÆMIA (typical of an irregularly permitting pyrexia).—Catherine W ——,
æt. six, admitted to hospital, September 27, 1881. She was taken ill somewhat suddenly on
September 13 with shivering and vomiting. On admission she was in a condition of prostration.
There were no physical signs excepting a systolic bruit over the whole cardiac area, and slight
enlargement of the spleen. On the 30th there was rusty sputum with streaks of blood; dulness
and crepitations over the right back. She was delirious from time to time, and died somewhat
suddenly on October 3. At the autopsy pus was found in the mastoid cells and sinus throm-
bosis secondary to long-standing middle ear disease (of which a history was now obtained), infarcts
in the kidney, and pyo-pneumothorax secondary to rupture of one of the gangrenous-looking
abscesses of the lung.

Prognosis.—The *course* of septicæmia differs widely. Thus, on the one
hand, some cases of intense septic infection from a *wound* or parturition
run a rapid and fatal course of ten or twelve days, terminating in the
" typhoid state." On the other hand, cases in which apparently small
quantities of septic matter are constantly leaking into the general circula-
tion from some *internal* source may be indefinitely prolonged over many
weeks or months, the mind remaining clear the whole time. Such would
appear to have been the course of the disease in the patient referred to in

Fig. 102.   There is, in fact, no definite line to be drawn between the
acute septicæmia now under consideration and the *subacute* and *chronic*
septicæmia due to pent-up pus or ulceration described below (§ 414).
Acute pyæmia is a most serious and, if untreated, invariably fatal malady.
Death may occur either by the intensity of the poison (typhoid state),
asthenia, or complications.   The *untoward symptoms* are a very high
temperature, frequent rigors, or cerebral symptoms.   The most frequent
complications are (1) pneumonia, which invariably occurs in severe cases ;
(2) pericarditis or pleurisy, which usually becomes purulent, and peri-
tonitis ; and (3) suppurative inflammation of the spleen, liver, and other
organs, consequent on the infective emboli ; (4) malignant endocarditis.
Among the sequelæ in certain less acute cases which recover may be
mentioned a destructive form of arthritis.

Treatment.—The indications are (1) to remove the cause ; (2) to inhibit
the microbic toxin ; (3) to relieve the symptoms and maintain the strength.
(1) If the infection is derived from a wound or some accessible purulent
cavity— *e.g.*, an abscess, an empyema, acute necrosis, etc.—this should be
promptly laid open, drained, and treated by antiseptic measures.   Search
must be made for some internal cause—*e.g.*, appendicitis—and this should,
if possible, be dealt with.   (2) Thanks to the researches of modern
pathology, we are now in possession of an antistreptococcic serum, and
several cases are on record which have been rescued from death by this
means.   As previously mentioned (and see § 417), several different organ-
isms may produce the disease, and we must identify which is in operation
before we can employ the appropriate serum.   The most frequent, when
the source is some purulent focus or abscess, is a form of streptococcus.
The great variety of forms of streptococci has proved to be the chief
difficulty in the serum treatment for septicæmia.   It therefore usually
happens that the serum given is not antagonistic to the particular organism
in operation, and a polyvalent is more likely to be successful than a
monovalent serum.   Recently vaccines prepared from the organism
obtained from the patient's blood have been tried, with success if given
early.   (3) The administration of quinine in large doses has some control-
ling influence over the temperature ; antipyrin, antifebrin, and other
febrifuges are also used.   The internal administration of antiseptics
generally has not been found of much use.   Stimulants and concentrated
nourishment are called for (see also §§ 421 *et seq.*).

§ 414. **Subacute and Chronic Septic Conditions** (*e.g.*, **Abscess, Ulceration,
etc.**) also give rise to intermitting pyrexia.   The various clinical condi-
tions met with under this heading are due to the absorption of some septic
or toxic material into the circulation.   The possible sources of the sepsis
are very numerous, and may be grouped into two divisions—(*a*) ABSCESS
(or pent-up pus) ; and (*b*) ULCERATION (internal or external).   Clinically,
the former is more acute than the latter, and, indeed, the former might
be called subacute, the latter chronic, septicæmia.

    (*a*) ABSCESS (PENT-UP PUS).—Pus never forms in any part of the

body—*e.g.*, in the pleura (empyema), in the liver (hepatic abscess), or elsewhere—without the occurrence of "chills," "shivers," or "rigors," and an intermitting or remitting pyrexia. Before the clinical thermometer was invented, these shiverings (sometimes followed by sweatings) were the chief symptoms by which the formation of pus was identified. When there is fluid in the chest, for instance, and we do not know whether it is serous or purulent, the occurrence of shivering or sweating will often settle the question in favour of pus. The temperature in such cases presents much the same chart as that in tuberculosis, though it has not such regularly diurnal variations, and is more often accompanied by shivering or rigors. There are considerable lassitude, debility, pallor (though with a hectic flush on the cheeks), and more or less loss of flesh in course of time. Albumosuria is usually present, and is a valuable confirmatory symptom. The blood should always be examined, and the presence of leucocytosis with an increase in the proportion of polynuclear cells will afford strong confirmation that pus is present.

Causes.—Abscess or pent-up pus in any position may produce these symptoms, and careful search should be made for abscess of the liver, spleen, or other organs, pelvic cellulitis, caries of the spine or mastoid bone, appendicitis (Fig. 102), intracranial abscess, empyema, pyonephrosis, etc. Pain is the chief localising symptom, but it may be wanting. On giving free exit to the pus the pyrexia should rapidly subside.

(*d*) ULCERATION of an INTERNAL or EXTERNAL surface (including the conditions known as "Hectic Fever," Hepatic, and Urinary Intermitting Pyrexia) is always attended by some degree of intermitting pyrexia, running a more chronic course than the foregoing. This fever also differs from the last in the usual absence of definite rigors. Sometimes the shivering may not amount to more than "chills down the spine"—thought to be malaria, perhaps—and sweating which is hardly noticed. The morning temperature is normal, or almost normal, and it is raised one or two degrees some time during the day. Anæmia and failing health are always present, but here albumosuria and the blood changes just mentioned may be absent. This kind of fever, due to prolonged suppuration, and attended by chronic wasting, was formerly known (and is still among surgeons) as *Hectic Fever* (Greek, ἑκτικός "habitual"). When due to a discharging sinus—a sinus, for instance, connected with caries, or necrosis of a bone, or a bed-sore—the cause is obvious. But the condition may also be set up by ulceration of the intestines or any of the mucous membranes or internal passages—*e.g.*, the appendix (Fig. 102). It is called *Urinary Fever* when it arises from chronic ulceration of some part of the urinary passages—*e.g.*, when a stone is impacted in the ureter, or when the patient has urethral stricture, or there is ulceration of the pelvis of the kidney (pyelitis). This cause may be suspected if there be a history of renal colic. Similarly, *Hepatic Intermitting Pyrexia* (ulceration of the biliary passages) may be suspected if there be a history of biliary colic. When the ulceration, due to gall-stones, is situated in the *gall-bladder*,

both colic and jaundice may be entirely absent, and the patient complains of nothing but the " chills " (§ 266).

§ 415. The **rarer causes of Intermitting Pyrexia** are fully described elsewhere, and, with the exception of trypanosomiasis, need only to be mentioned here.

Influenza, Enteric, and **Paratyphoid** fever, especially when modified by previous inoculation, and other diseases described in Groups I and II may be attended by pyrexia of an intermitting type (§ 394).

Kala-Azar has usually intermittent fever after the first period, during which there is fever of a remittent type.

Enteric Fever during the first two weeks of its course is attended by typically continued pyrexia, but in the concluding stage of the disease the pyrexia gradually drops each morning to normal, and the case may be seen for the first time in this stage.
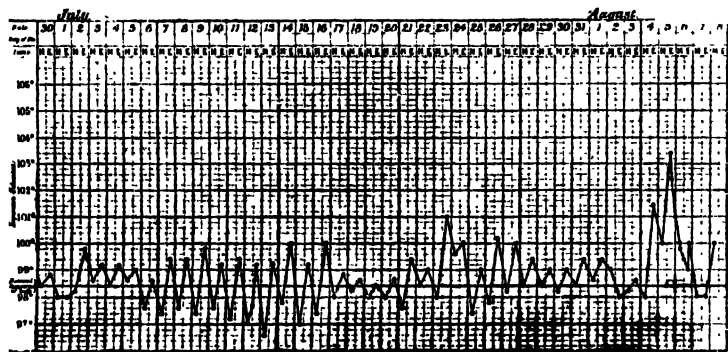
Fig. 102.—CHRONIC PYÆMIA.—Frank T——, æt. thirty-one, had had an attack of gonorrhœal rheumatism two years before, from which he had recovered. The present illness had come on quite gradually a month or so before admission. Stiffness and pain in the joints being the chief symptoms, and the urethra being *absolutely normal*, it was regarded as a case of chronic rheumatism, though none of the usual remedies had any effect. The joints became progressively worse, and though he complained of abdominal pain from time to time attention was not directed to that cavity. He died some two months later suddenly from perforation of the appendix vermiformis. A review of the case pointed to a chronic septic process having its origin in the appendix, and specially affecting joints which had been previously diseased.

Under certain other circumstances also the temperature may be intermitting—viz. : (i.) In rare instances it may commence with symptoms of ague (Murchison); (ii.) in very mild cases the temperature may be intermittent; (iii.) after lasting a few days, the fever sometimes aborts and takes on an intermitting type. For the diagnosis of the disease we now have valuable guides in Widal's test and a blood culture.

Various **local inflammatory diseases,** other than the septic conditions previously mentioned, may at times be attended by intermittent pyrexia. In cirrhosis of the liver, for instance, a prolonged fever with daily oscillations has occasionally been observed.

Malignant Endocarditis (Multiple Systemic Embolism) (§ 46) is always attended by pyrexia of an irregularly intermitting type, sometimes with sweatings and rigors, very much resembling the chart of septicæmia, though the temperature is usually a little more diurnally regular, and rigors are not usually so frequent (compare charts, Figs. 101 and 103). The diagnosis of these two diseases is sometimes very difficult (§ 46). Malignant Endocarditis is favoured by (i.) the existence of a loud cardiac murmur detected quite early in the case ; (ii.) a history of acute rheumatism ; (iii.) the secondary emboli in this disease are more frequently found in the systemic arteries,

such as those of the spleen, liver, and kidneys, and they do not result in abscesses. In pyæmia the emboli occur primarily in the arteries of the lungs, and from the very beginning they suppurate and form abscesses, which constitute centres of secondary infection elsewhere.

Hodgkin's disease is recognised by the enlargement of the lymphatic glands. This enlargement is attended by pyrexia of an intermitting character (§ 438).

In **Pernicious Anæmia** the temperature is sometimes subnormal, but it is more frequently attended by exacerbations of fever of an intermitting type. Rigors and sweats may also occur, but they are not usual. The disease is also identified by the intense sallowness of the skin and the condition of the blood (§ 433).

In **Acute Lymphatic Leukæmia** the temperature is high and irregular, somewhat resembling that of septicæmia. It can be diagnosed by the examination of the blood, when there is found to be an increase in lymphocytes (§ 437).
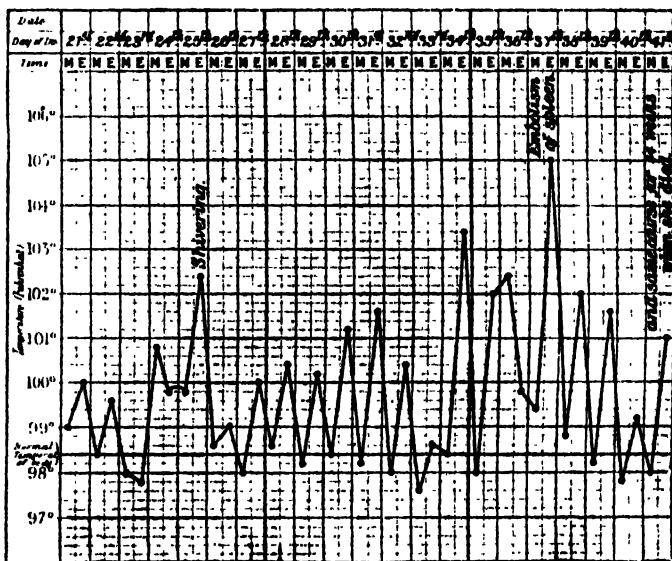
FIG. 103.—MALIGNANT OR ULCERATIVE ENDOCARDITIS in a female patient, æt. forty-two, who was admitted to the Paddington Infirmary in the year 1890. The three weeks shown illustrate the course of the temperature over a period of seventeen weeks, when she died. The chart of another case will be found in § 40.

The **Opium or Morphia Habit** is attended from time to time by attacks of intermittent pyrexia, during the reaction stage, in which there are cold, hot, and sweating stages. Dr. Livenstein calls attention to this fact, and records cases where no other cause could be found, and where the attack ceased on giving opium.

Trypanosomiasis (Synonym : Sleeping Sickness) is a disease occurring in various parts of Africa, characterised by enlargement of the glands, often an erythematous rash, irregular pyrexia and excessive sleepiness.

Symptoms.—Three stages are recognised. In the first there is enlargement of the glands in various situations, especially in the posterior triangle of the neck. In many cases, especially when the disease affects Europeans, an erythematous rash is present which becomes ring-, then crescent-shaped. The eruption often coincides with irregular pyrexial attacks. The second stage may last on and off for two to three years, with symptoms of hectic fever, increasing lassitude and disinclination to work, anæmia and wasting. The third or final stage lasts several weeks. There is

increasing lethargy ; the intellect is dull, the face puffy, the gait shuffling ; there are tremors of the tongue, lips and limbs. The temperature is high at night ; normal in the morning. Drowsiness increases to profound lethargy, and ends in coma and death.

Etiology.—The disease follows the bite of a tsetse fly, by which the trypanosoma is introduced into the body. There are two varieties ; that form prevalent in Uganda is very widespread and epidemic ; the form met with in Rhodesia and Nyasaland is less widespread, is apparently not epidemic, but is more rapid in development and fatal. The *Glossina palpalis* carries the Uganda disease ; this tsetse fly never goes far from water. In Rhodesia and Nyasaland the *Glossina morsitans* conveys the disease ; it is independent of water. The part played by wild animals in the dissemination of sleeping-sickness is not yet clearly known.

The *Diagnosis* can only be made with certainty by finding the parasite in the blood or cerebro-spinal fluid, or by puncturing one of the enlarged glands. The last is the most, and the first the least, valuable method to adopt.

The *Prognosis* is very grave. If the patient has not shown any signs of the third stage, if he can leave the country and have vigorous treatment, the prognosis is better than otherwise, but is not good.

Treatment.—A valuable drug is Atoxyl or Soamin ; 2 3 grs. (0 12–0 2) should be given intramuscularly every third day for at least two years. Bayer 205 is giving even better results.

Chaga's disease in S. America is also due to a trypanosome.

Trichinosis is usually accompanied by intermittent fever.

Rat-bite fever may show a temperature of an intermittent type.

THE GENERAL TREATMENT OF MICROBIC DISORDERS

Remedial treatment has, for the most part, been given under each disease, but there are some important matters relating to all fevers in common which must now be referred to—viz., **Immunisation, Serum Therapeutics, Notification** and **Isolation, Disinfection, Diet,** and the treatment of **Pyrexia** and **Hyperpyrexia.** In the first two of these we find ourselves on the threshold of discoveries which are revolutionising the methods of treatment and prevention of infective disorders.

§ 416. **Immunity** signifies non-susceptibility to a disease, a poison, or an organism. The term has been given a much wider significance, and includes the entire subject which deals with the variation of susceptibility of various peoples, persons, animals and plants to the many poisons by which they may be attacked. In this connection it is as well to leave out of consideration the resistance which may undoubtedly be developed to simple chemical poisons. Though the Styrian mountaineer may be able to swallow unharmed a dozen fatal doses of arsenic and the opium addict be able to drink laudanum by the wineglassful, the mechanism is entirely different from that by which immunity to the more complicated poisons of bacteria is produced. The nature of these various poisons will be briefly outlined.

Toxins.—The poison produced by bacteria when infecting the body is known as a " Toxin." Its behaviour in the body is comparable to snake poisons on the one hand, and on the other to the poisons produced by certain plants, *e.g.*, ricin, the poison in the castor oil bean. Quite early

in the history of bacteriology it was found that some pathogenic organisms produced during their growth a soluble poison which could be obtained, free from all bacteria, by filtering the culture through a Chamberland candle of suitable size. The toxin so obtained is known as an " Exotoxin," being outside the bacterial body. The Corynæbacteria Diphtheriæ, the Tetanus and the Botulinus bacillus are well-known examples of the organisms forming an exotoxin. These toxins when injected into susceptible animals in suitable doses produce all the symptoms of the original disease. On the other hand, the vast majority of organisms, even the most consistently pathogenic, produce in their cultures no trace of toxin whatever. Nevertheless, if the organism is killed, or frozen and minced, the injection of the dead bodies of the organism will in many cases produce lesions more or less comparable to those found in the original disease, though more often it is the general effect rather than the particular local lesion which is produced. From this it is concluded that though no exotoxin is formed, there is some toxic substance within the organism which produces its due effect even though the organism is dead. This is known as " Endotoxin."

The nature of both an Exotoxin and an Endotoxin remains obscure. It is known that toxins may be passed through a very small porcelain filter, that they are associated with an albumose, and that they cannot be crystallised, which suggests that they are some colloidal substance ; but whether they are the albumose, or only some substance absorbed on its surface is not known. Whatever their nature, their potency is almost incredible ; the fatal dose of pure tetanus toxin for a guinea-pig has been estimated at 0·000001 gramme, whereas the fatal dose of strychnine for a similar animal is more in the neighbourhood of 0·01 gramme. The potency of all toxins decreases with age, and may be reduced and finally destroyed by heat. A similar effect may be obtained by chemical means, and also by such treatment as shaking.

Toxins are not only classified from the point of their origin in the cell, but also from their mode of action in the body. Thus those which attack the phagocytes are called " leucocidines " and " aggressins " ; those which attack the nerves are called " neurotoxins," and so forth. The most characteristic effect produced by this type of poison, and that which distinguishes it from the simpler poisons is the development by the body of immunity towards them. When a toxin is injected in a non-lethal dose it causes the body to develop a certain degree of resistance to that particular toxin, and by repeated doses of the toxin this immunity can be developed to a remarkable extent. It is further found that this immunity, which has become a property of the first animal, can be transferred to a second animal by injections of a suitable quantity of its blood or serum. The substance developed in the animal, which thus neutralises the toxin, is known as the antitoxin. From the power which toxins possess of producing antitoxins, they are classified as " antigens." A pure antitoxin has never been obtained, and our knowledge of their chemistry

is as vague as is our knowledge of that of the toxins. The antitoxins have a larger molecule than have toxins, for they may be filtered out of a solution by a filter which allows the passage of a toxin; they are more stable as regards age, less stable as regards heat, and are probably proteins.

With these facts in mind, we are in a position to approach the most important aspect of the subject, *i.e.*, the development of immunity in the animal body. The primary division of immunity is into two classes: natural and acquired.

Natural immunity is the inherent resistance of certain animals to certain toxins, for instance hens to tetanus toxin. But even in this case the immunity is only relative, not absolute; given a sufficient dose of tetanus toxin, even a hen will succumb. So also sheep, which live an open-air life, are as a rule immune to the tubercle bacillus; but when they are kept penned in, in insanitary conditions, they may become infected. On the other hand, a natural immunity may often (though not always) be enchanced by suitable treatment.

Acquired immunity is the resistance developed to a toxin as a result of natural disease or artificial treatment. It is a matter of common knowledge that some diseases rarely attack the same person twice, *e.g.*, typhoid, scarlet fever and small-pox. Here again the immunity is relative and not absolute; second and even third attacks have been described. Artificially acquired immunity is the crux of this subject, and is the line of research along which immense progress has been made, and even more is promised. Inoculation of small-pox is probably the earliest example. The serum from a small pustule in a mild case was rubbed into the skin, and a mild attack of small-pox ensued. The next step was that of Jenner, who, observing the immunity to small-pox of those already infected with cow-pox, tried and succeeded in immunising people against small-pox by artificial vaccination with cow-pox. Whether cow-pox is an allied disease, or a modified form of small-pox is still a subject for argument, but quite immaterial from the point of view of immunity. After this discovery little progress was made till Pasteur began his work on hydrophobia and anthrax, which may be considered the starting-point of our knowledge of the now vast subject.

Artificial immunity is divided into two classes, Active and Passive. In *Active immunity* the resistance is developed by the body as a result of the injection of the toxin, or organism or virus. Vaccination is therefore an example of active immunity. In other conditions it is developed by injecting doses of the poison graduated in strength, beginning with weak and working up to strong doses. The modification of the strength is variously obtained, dead bacteria may be used, or old or overheated toxins, or the bacteria may be grown on unsuitable media, such as one containing disinfectants, or at an unsuitable temperature, or in the absence of oxygen or in excess of it. Minute doses only may be used or the appropriate anti-serum added. Later, larger doses or more toxic strains are employed,

and finally it may be necessary to give large doses of living virulent bacteria, or fully potent toxin.

In *Passive Immunity* the serum from an animal which has been actively immunised against the poison in question is injected into the subject, thus effecting an almost immediate increase in the patient's resistance to the poison.

While the observed facts of immunity are clear and definite, the theoretical explanations of the problems are far less satisfactory. It is most important to remember that the theories are only explanations, and are valueless if they cease to provide a satisfactory basis for further research and speculation. As regards the neutralisation of toxin by antitoxin, three theories at present hold the field ; not one is completely satisfactory. Ehrlich's theory is based on the presumption that toxin and antitoxin unite as do strong acid and strong base. However, the discovery of the fact that with age a toxin loses its toxicity, but will still unite with the same quantity of antitoxin, necessitated the introduction of a " toxone," a substance allied to a toxin ; from this point there have been continual modifications of the original theory to meet newly discovered facts. The explanations are nearly as complicated as the facts they set out to explain—an obvious disadvantage. Nevertheless, this theory has been the starting-point of the greatest amount of work on the subject. Madson and Arrhenius have suggested that the combination of toxin and antitoxin is comparable to " mass action " in chemistry, on the analogy of the combination of a weak acid and base ; the theory has proved helpful, but does not cover all the possibilities. Lastly, Bordet, forsaking chemistry in its narrower sense, has suggested that the union is of the nature of adsorbtion, the physical electrical interaction of substances in the colloidal state, and this theory, which makes the least hard and fast rules, seems to be the most fully observed.

To account for the almost unlimited production of antitoxin which may follow the injection of comparatively small doses of toxin Ehrlich offers the following suggestions : Toxin and body cell consist (from the chemical point of view) of a number of complicated organic substances or " side chains," loosely attached to a central nucleus. A toxin can only exert its poisonous properties when one of its own side chains unites with the appropriate side chain of the cell. This union is permanent, and so puts an end to the vital activities not only of the single side chain but also of the whole cell ; when this process takes place extensively, death results. In the case of a non-fatal dose of toxin, the cells ultimately recover ; as they recover they regenerate the side chain neutralised by the toxin. The number of side chains regenerated not only replaces those destroyed, but provides an excess ; as the cells do not need them they are shed into the general circulation and form the antitoxin. As a result of repeated injections the body not only produces a great quantity of antitoxin, but develops the power of developing even more, and that without the stimulus of a further injection of toxin. An immunised

animal may be bled repeatedly, till the total volume of blood lost is in excess of its total blood volume and yet the quantity of antitoxin in the new formed blood (*i.e.*, its antitoxin titre) will equal if not exceed that of the blood before the depletion. The practical application of our knowledge of immunity in the treatment of disease must now be briefly considered.

§ 417. REMEDIAL IMMUNISATION.—**Vaccine Therapy** is based on the principle of producing in the patient a condition of active artificial immunity, and various methods are employed, all leading to this end. The most usual is the injection of suspensions of the dead bodies of the infecting micro-organisms. Sir A. Wright and Captain S. R. Douglas, while studying the properties of phagocytosis by the leucocytes, originally described by Prof. Metchnikoff, found that the proportion of organisms ingested by the polymorphonuclear leucocytes was increased in tests where the blood of persons who had received injections of the organism in question was employed. Further investigation showed that this property of phagocytic increase was situated in the serum, for it did not occur in tests with washed leucocytes. On the other hand, it was found in tests with washed leucocytes from a normal person under treatment with the serum of an inoculated individual. The substance which was the cause of this phenomenon they called *opsonin*. In the first instance the aim of vaccine therapy was to increase the "opsonic index" of the patient's blood, and the estimation of the opsonic index is still the most satisfactory means of estimating the patient's
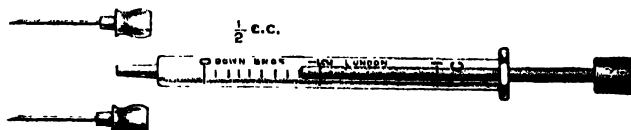
FIG. 104.—EYRE'S VACCINE SYRINGE.

immunity, and its progress with treatment. The actual immunity, however, depends on many substances of which opsonin may be taken as a type : agglutinins, precipitins, bacteriolysing and bacteriocidal bodies. In constitution a vaccine is a suspension of organisms in fluid. From the infecting focus a culture is made, and from this culture the various types of organism are picked off and examined. Pure cultures of each pathogenic type are taken, then rubbed up in normal saline and a homogeneous suspension made. This is then standardised either macroscopically, by comparing its opacity against that of a standard suspension ; or microscopically, by comparing, in a stained film made from equal quantities of the suspension and of blood, the number of organisms and of red blood corpuscles. The doses of most vaccines are calculated in millions of organisms per cubic centimetre, and may be put up in separate ampoules, or kept in bulk in rubber-capped bottles.

Sensitised vaccines are similarly prepared, but before standardisation the suspension of organisms is treated with the serum of an animal, or a person immunised against the organism in question. After about 12 hours, the organisms are washed free of the serum, diluted with tricresol water, standardised, and bulbed in the ordinary way. Live organisms are sometimes used. *Detoxicated vaccines* are made by dissolving or otherwise destroying the cell wall of the organism. It is claimed that they produce less reaction, that larger doses are possible, and that better results are obtained. Diaplete, *defatted vaccines* have been recommended by Professor Dreyer ; in these the cell wall is rendered less resistant to absorption by the removal of its fats. It is not possible to give even a list of the many varieties of vaccines which have their respective advocates.

When injecting vaccines, the following procedure should be adopted : (1) The

skin should be sterilised with a dab of ether or iodine; (2) The syringe, preferably an all-glass one, should be boiled; the needle may be boiled, or sterilised in spirit, ether, lysol, or boiling oil; (3) The site selected should be on the body. Below the clavicle, over the scapula, or on the abdomen are very convenient places. It is better not to select the arm for the site of injection, as in the event of a marked local reaction, the swelling might be very painful and free drainage is more difficult. It is usual to give vaccines in progressively increasing doses, and to allow an interval of at least a week between the smaller doses, and of ten days or a fortnight between the larger ones. The beneficial results depend very greatly on the exact relationship between dose and interval, for the immunisation aimed at has to be developed by the patient, and is not spontaneously conferred by the injection of a dose of vaccine. In this respect it is specially important never to begin a vaccine at the time of a woman's menses, even the late doses should be held over if possible.

§ 418. SERUM THERAPY.—In certain diseases, or when it is desired to develop immunity in the shortest possible time, or when a vaccine is unsuitable, it is possible to confer passive immunity on the patient by injecting an antitoxin. This consists of the serum of animals which have been immunised to the organism in question. In some cases the doses are calculated in arbitrary standard units; in others, in terms of volume. The bulk of serum employed may be considerable.

Serum may be given by four routes : (1) Intravenous. This is the method of
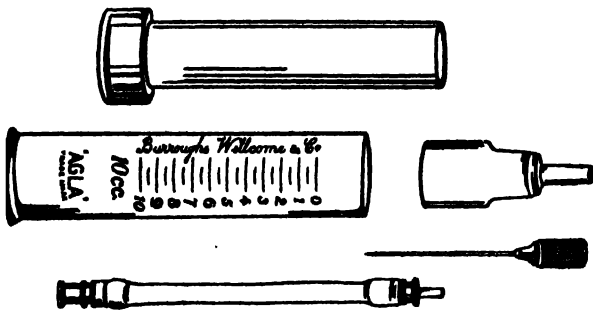

FIG. 105.—ALL-GLASS SYRINGE.

choice when immediate immunisation is required, for the antitoxin is thus instantly available. It is also the best way when doses have to be large, or frequently repeated, as in dysentery or pneumonia. On the other hand, the immunity is less lasting, and there are certain dangers which will be mentioned later. (2) Intra-muscular. The glutei are the most convenient muscles. This method is less rapid in its effects, the maximum concentration of antitoxin not being present till twenty-four hours after the injection. It is, however, much safer, and requires less elaborate technique. (3) The subcutaneous method of injection is the slowest and the easiest. The maximum concentration is not reached till seventy-two hours after injection. The injection may be made in the flanks, abdomen or thighs. This route may be of great value as a reserve in support of the intravenous, the antitoxin being maintained in the circulation by slow absorption. The combined administration of antiserum and vaccine by subcutaneous injection is very effective, especially in the case of infection by organisms of the streptococcus type, where the serum alone is frequently of small avail. (4) Intrathecal. In the treatment of tetanus and cerebro-spinal meningitis this route is invaluable. A lumbar puncture is performed and cerebro-spinal fluid is drawn off in excess of the quantity of serum which is afterwards injected. A Record or all-glass syringe must always be used (Fig. 105).

SERUM-SICKNESS.—The injection of any serum may be followed in eight to twelve days by a series of definite symptoms, the most common being an urticarial rash

which, starting at the site of the injection, may extend over almost the whole body. Enlargement of the lymphatic glands, either local or general, may occur. The temperature may be raised, and pain and swelling of the joints accompany a still more severe type. In the most serious cases there are definite signs of nephritis. The symptoms invariably subside, but may persist for as long as five weeks. Sudden collapse during, or immediately after an injection is rare, and is most common after intravenous injections ; fatal cases are not unknown. True anaphylaxis does not seem to occur in human beings, and it is usual to ascribe serum sickness to allergy. Though the condition is not true anaphylaxis, serum sickness is more common after the second or third dose of serum than after the first injection. It is also more frequent after intravenous than after either intramuscular or subcutaneous injection. It is therefore advisable to reserve the intravenous route for urgent cases only, and, even then, to give a minute dose, say 0·5 c.cm. half an hour before the main injection ; the same precaution may well be taken in second, third, or subsequent injections, whether by the intravenous or other method. Large doses of calcium lactate are also recommended to be given in advance ; thirty grains three times a day. For the acute collapse the usual restoratives should be employed, and adrenaline would possibly be helpful.

ALLERGY is defined as a natural inherited condition of hypersensitiveness. Serum sickness is one manifestation of allergy ; it probably explains a number of other conditions. Of these the best known and the most common, is Hay Fever, the result of hypersensitiveness to the pollen of Timothy grass. Under this heading also comes. the hypersensitiveness shown by certain people towards various foods, such as strawberries, eggs, shell-fish and the flour of various grains ; and after proximity to certain animals, *e.g.* the horse and the cat, and to certain plants and flowers. Various symptoms are produced ; food stuffs as a rule cause urticarial rashes ; animals and flowers, asthma. Complete sets of common proteins are available for testing purposes. The method employed is to scarify the skin and to apply the substance to be tested to the scratch. In from 1 to 10 minutes a reaction appears which varies in its intensity with the sensitiveness of the patient. The number of substances now known to produce the condition of allergy is legion.

SPECIAL METHODS FOR EACH DISEASE.

I. DIPHTHERIA.—An antitoxic serum has been in the market since 1895. When given early enough and in large enough doses, it has been found to be of the greatest value as a remedial agent for patients suffering from the disease (see Comparative Mortality, § 416). The *Contra-indications* for its use are given in § 392.

Method.—The remedy should be used as early as possible in the disease. A dose of 4000 to 8000 units should be given, and repeated in half-doses every twenty-four hours, or every twelve hours if the case be very severe, until the exudation is obviously separating. Avoid injecting more than 20 c.c. at one place, if possible. The usual site for injection is subcutaneously in the flanks. It is very important that the injection should be made early ; the earlier the antitoxin is given, the more favourable is the prognosis. Therefore, in suspicious cases, where a bacteriological report cannot be got at once, the correct treatment is to inject the antitoxin without waiting for the report. Children tolerate antitoxin well, and should receive the same doses as adults.

In addition to the known curative results which are obtained by the use of the diphtheria antitoxin it is now possible to obtain a lasting prophylactic action by means of a combined dose of toxin and antitoxin. This " Toxin-antitoxin " mixture is adjusted to contain a slight excess of toxin over antitoxin ; three injections at fourteen to twenty-one days' intervals are recommended. Such a course appears not only to convert a " Schick-positive " patient to a " Schick-negative " one, but also to confer a real and lasting immunity.

Effects.—In the course of twenty-four hours there should be an improvement in the patient's symptoms : the membrane ceases to extend, or perhaps begins to loosen, the swelling abates, and the rhinorrhœa is diminished. Occasional effects are urti-

canal or erythematous eruptions, additional rise of temperature, or joint pains and swelling.

TETANUS (Fig. 106).—The mortality from tetanus in our armies has been practically arrested since prophylactic injections of 500 U.S.A. units have been administered as a routine measure of precaution as soon as possible after a man has been wounded. The first dose loses its effect in about ten days; therefore the practice advised is to repeat the injections every seventh day for three or four doses. If the disease appears, 5000 units should be given at once; 20 c.c. of cerebro-spinal fluid should be withdrawn and 20 c.c. antitoxin injected every third day. At the same time 5000 to 10,000 units should be injected intramuscularly and subcutaneously in the neighbourhood of the wound.

Some ten days later an urticarial rash is common near the site of the prophylactic injection, and widespread symptoms of the "serum disease" may occur (§ 418 and § 485).

SEPTICÆMIA and pyæmia (including Erysipelas, Malignant Endocarditis, and Puerperal Fever).—The pyogenic (pus-producing) organisms are capable of producing septicæmia—streptococcus, staphylococcus, B. coli communis, etc.—and the antiserum of one will not act upon another. Anti-streptococcic serum may be tried if a streptococcic infection is in operation.

Method.—As in all cases treatment should be commenced early, and since the case may be one of mixed infection, some do not consider it advisable to wait for a bacteriological report. Start with 20 c.c., and repeat once or twice daily as long as it appears to reduce the pyrexia.

. Effects.—In successful cases there should be an almost immediate fall of temperature and improvement in the condition of the patient.

Vaccine Therapy.—A culture is made from the patient's blood, and a vaccine prepared from it. Successful cases have been reported.

FIG. 106.—TETANUS BACILLUS.—× 1000. Cover-glass preparation. Gentian violet. Photomicrograph by Mr. Frederick Clark.

The course of surgical infections of all varieties, appendicular abscess, osteomyelitis, empyema, and septic arthritis, is frequently accelerated by the exhibition of autogenous vaccines, supplementing the rational surgical treatment of the condition. Unfortunately such conditions as Septicæmia and Malignant Endocarditis show as little response to this as to any other form of treatment at present known.

Vaccines are used both for prophylaxis and the treatment of many diseases. A list of the more common is given below.

ANTI-CATARRHAL Vaccine.—This is composed of suitable doses of all those organisms which may infect the nose, mouth, throat and lungs. These are the Streptococcus Longus and Streptococcus Viridans, the Pneumococcus, the Pneumobacillus, the Influenza Bacillus, Micrococcus Catarrhalis, and various strains of Diphtheroid Bacillus. It is usual to give this vaccine in three doses, increasing the number of organisms at each dose. These are given at weekly or ten-day intervals. The protection bestowed lasts from one year to two or three years.

TYPHOID and the PARA-TYPHOIDS (Fig. 107).—The original anti-typhoid vaccine has now been almost entirely replaced by a mixed vaccine containing Bacillus Typhosus and both Bacillus para-typhosus A and B. This is the vaccine known as T.A.B., which was used with such success in all theatres of the war. Two injections of 500 million and 1000 million respectively are given at ten-day intervals. Protection lasts for one or two years.

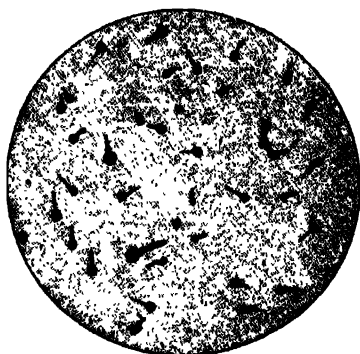TUBERCULOSIS.—(a) Koch first introduced a "tuberculin" made by filtered

bacillus cultures. This product is now known as the " old tuberculin," and is used solely for diagnostic purposes, for it only produces a reaction in an individual when he has tuberculosis in some part of his body. In this country it is used chiefly for animals.

Method.—Half a milligramme is injected, and the temperature is taken every four hours. If during two days there is no rise, 2 milligrammes are given, and the temperature taken for two days again ; then 5 milligrammes, and the temperature again taken. If there is no elevation of temperature at any time the case is not one of tuberculosis.

(β) Koch next introduced a new tuberculin (T.R.), which consists of finely-powdered bacilli, washed free of toxins.

(γ) Lastly, he introduced a bacillary emulsion (B.E.), a suspension of finely-powdered bacilli in water and glycerine. This is rendered sterile by heating to 60° C., and is now generally employed. The dosage of all the tuberculins is a matter on which there are great diversities of opinion. Those usually recommended range from $\frac{1}{50000}$ milligramme to $\frac{1}{5000}$ milligramme of either the T.R. or the B.E.

HYDROPHOBIA.—The Pasteur treatment of hydrophobia has obtained a world-wide reputation. Rabbits are inoculated with the virus of hydrophobia, and their spinal cords are taken out and dried. The longer these are allowed to dry, the more attenuated is the virus contained by them. Emulsions are made of the cords, and these are injected into the patient. Weak cords, which have been dried for fifteen days, are first employed, and the virulence of the cord employed is gradually increased for ten days.

Indications. — Treatment must be commenced as early as possible after the date of infection. The danger to be avoided in the treatment is a too rapid increase in the strength of the virus.

FIG. 107.—TYPHOID BACILLUS, × about 1000.

On no account should the dog be killed if this can be avoided. It should be caught and kept under observation. If it does not die, it is not rabid, and there is no danger of hydrophobia ; if it does die, the patient should hasten to the nearest Pasteur Institute with the dog's body packed in ice. At the Pasteur Institute the necessary treatment can be applied. The immediate treatment of the wound is cautery, the more thorough the better. The results of the Pasteur Institute method of treatment are very encouraging. The ordinary mortality of bitten patients, before the institution of this treatment, was about 16 per cent., but from 1886 to 1895 (17,337 cases) the mortality was 0·48 per cent. A serum treatment of hydrophobia is at present on trial.

PLAGUE.—Both a serum and a vaccine are employed as preventives for this disease. The serum gives an immediate but transient protection, while the vaccine takes longer to work, but is more lasting in its effects.

Contra-indications.—The treatment must be commenced on the first day of the illness, because the course of the disease is so rapid and severe that later administration cannot check its progress.

Method.—From 20 to 40 c.c. are injected daily for one to ten days, according to the nature of the case.

Effects.—Cases have recovered in two days when the treatment was commenced on the first day of illness. In such cases the mortality has been greatly reduced.

, CHOLERA.—A vaccine containing 12,000 million cholera bacilli to the cubic centi-

metre has proved a valuable prophylactic in epidemics of this disease. The protection afforded being only for four months or so, it is of less value than T.A.B. in enteric. It is most satisfactorily employed during an epidemic, when all persons in the infected area may be immunised, which immunity will last till the emergency has passed.

SNAKE POISON.—Calmette introduced an antitoxic serum for the poison of snake bite, which is known as "antivenene." It is effective against the venom of the colubrine snakes, but often fails to neutralise that of the viperine species. The serum can be kept for a long period in a tropical climate without losing its properties.

Method.—Inject as soon as possible after the bite at least 10 c.c. of Calmette's antivenene, and repeat the dose some hours later.

Effects.—The patient recovers very soon if the injection is given before unconsciousness or paralysis set in. Even if given when respiratory paralysis threatens, this dangerous symptom may not ensue, and the paralysis of the limbs usually disappears in less than two days.

PNEUMONIA.—The serum treatment of Pneumonia has received a renewed impetus since the work on the grouping of the Pneumococcus has become known. Cultures in broth, or organism-laden peritoneal fluid from an inoculated mouse is tested as to its precipitin reaction against anti-pneumococcal serum of the four groups, and the appropriate serum is then used. This method has been successful in America.

Vaccines in some hands have given excellent results; both stock and autogenous have been employed (Cp. § 100).

ANTHRAX.—Animals have been inoculated with increasingly virulent doses of anthrax cultures, and the results are encouraging as a preventive. Sclavo's anti-anthrax serum has given good results in man. Inject 30 to 40 c.c. subcutaneously distributed in several situations, and repeat in twenty-four hours; in severe cases, inject 40 c.c. intravenously as well.

CEREBRO-SPINAL FEVER.—Flexner has introduced an antibacterial serum. This is injected into the spinal canal on several successive days in doses of 15 to 30 c.c. The results are excellent. It should be used in all cases of this disease.

BACILLARY DYSENTERY.—Very satisfactory results have been obtained by the use of a polyvalent serum. Large doses may be given intravenously; for example, 40 c.c. on the first day, followed by 50 c.c. and two doses of 60 c.c. on succeeding days. Owing to the toxic nature of the Shiga type bacillus it has not been found possible, in spite of many attempts, to make a really satisfactory prophylactic vaccine for this condition.

ULCERATIVE COLITIS also reacts very well to serum in some cases. The serum employed should be determined as a result of the bacteriological examination of the stools. Anti-dysenteric and anti-streptococcal sera may be used when there is doubt as to the causal organism.

Curative vaccines are employed, with results which vary between highly successful and absolutely negligible, in the treatment of all diseases for which a causal organism of the nature of a Bacterium has been isolated. Many of the failures of vaccine treatment are due to the fact that it often happens that vaccines are only tried when all other treatment has failed, and it also sometimes happens that vaccines are tried alone, with none of the other forms of treatment usually found effective.

Infections of the SKIN with the Staphylococci or with Streptococcus Longus, as boils, carbuncles, impetigo, pustular acne, and some types of eczema, react very well to vaccines combined with the orthodox local and general treatment.

Infections of the RESPIRATORY TRACT, such as chronic catarrh, chronic pharyngitis, certain types of bronchitis, bronchiectasis, asthma, and secondary infections of tuberculous cavities, react very well to autogenous vaccines combined with medical treatment. The infecting organisms may be Streptococci, Pneumococci, Pneumo-bacilli, Influenza bacilli, etc.

In RHEUMATOID ARTHRITIS, especially in early cases, but in all cases where the joint is not entirely destroyed beyond all hope of repair, vaccines prepared from the organisms isolated from the infecting focus are of the utmost use. The removal, or

cleansing, of the focus itself **is**, of course, the first step in the treatment. This focus should be sought not only in the teeth, but also in the tonsils, the accessory air sinuses, the lungs, the alimentary canal, and the genito-urinary system, and no examination can be counted as complete unless all these have been investigated.

PYORRHŒA ALVEOLARIS.—In this condition teeth may often be saved by the use of a vaccine coupled with conservative dental treatment.

CYSTITIS.—Chronic infections with the Bacillus coli react well to autogenous vaccines. Very small doses, ¼ to 1 million, should be used to start with.

§ 419. Notification and Isolation.—Two duties are laid upon the medical practitioner in cases of the commoner infectious maladies : (1) NOTIFICATION of the case to the medical officer of health of the district in which the case arises. The notifiable complaints in most districts are scarlatina, diphtheria, " membranous croup," poliomyelitis, enteric fever, and " continued " fever, small-pox, cholera, erysipelas, typhus, relapsing fever, phthisis, puerperal fever, and plague. To these acute poliomyelitis, varicella and cerebro-spinal meningitis have recently been added. Measles and whooping-cough may at any time be added to the list at the option of the Sanitary Authority. A medical man is bound, under a penalty of forty shillings, to notify any of the maladies named "immediately on becoming aware " of its existence. (2) REMOVAL of the patient to a fever hospital is compulsory, unless the parents or guardians can make *proper* and *adequate* arrangements for the isolation of the case at home. In some places the removal is superintended by the medical officer of health. In the metropolitan area the medical practitioner should at once communicate with the central office of the Metropolitan Asylums Board, Victoria Embankment, E.C., when an ambulance will promptly be sent for the case. Their telegraphic address is " Asylums Board, London," and the particulars required to be sent are Name, Address, Disease, Age, and Sex of patient, and Severity of case.

It is far better for the patient and for his relations that he should be removed to a properly organised Fever Hospital ; but to isolate a patient at home, hang a sheet, constantly wet with carbolic solution (1 in 20), across the door or passage. Carpets, curtains, and superfluous furniture should have been previously removed. Books and articles in use must be such as can be afterwards burned. Ventilation must be carried out as described below. The nurse in charge of an infectious case should wear a washable dress when on duty, and should hold no communication with others, nor should she go out of doors without having first changed her wearing apparel, and, if possible, taken a bath. An airy, quiet room *at the top of the house* having cubic space of about 12 × 12 × 10 feet, is desirable. The air ih this space requires to be changed three or four times in every hour. Only the furniture in immediate use should be allowed to remain. The carpet should be taken up, and all stuffed furniture removed. The bedstead should be so placed as to be accessible on both sides. The temperature, read on a thermometer suspended near the bed, and away from draughts, should be 60° F.

VENTILATION must be ample in fever cases, because of the danger of mixed infections. There are reasons for believing that the tonsils are sometimes the portal for infection, and that, perhaps, is the reason why mixed infections are more apt to arise in cases of scarlatina when there is not free ventilation and sufficient cubic space. This partly explains the higher death-rate from infectious diseases when overcrowding occurred in former days. The direction of the wind should be constantly noted, and to avoid draught, the windows or ventilators opened on the side of the room away from the wind. A " sash-board " is an excellent contrivance for avoiding draught. It should be about 6 to 8 inches broad, and fit across the bottom of the window, so that the lower sash can be raised without a visible opening, and then ventilation takes place behind the sash-board, and also *in the middle* of the window, the air in both cases being directed upwards. The chief principle involved in all ventilation is that the current of air always takes place from a colder to a hotter medium—usually, therefore, from outside to the inside of a room. The chimney, when the fire is alight, is the only reliable *exit*. Make the window your *inlet* in preference to the door.

§ 420. Disinfection and Prevention.—Before describing the means employed for disinfection, it is necessary briefly to describe the way in which microbic disorders are propagated. Since bacteriology has become a science, great advance has been made in this direction. There are three principal ways by which infection is conveyed—by the *air*, by *water* or other ingesta, and by direct *contact* or inoculation—and microbic diseases may be thus classified. ·,

(*a*) As regards the *air-borne* group, there is considerable variation in their infectivity, also the distance to which the contagion in an active state may be carried through the air. For instance, erysipelas and typhus probably do not spread beyond a few feet, but small-pox and scarlatina may spread for many yards ; some say the former spreads to a distance of a mile or more.[1] Air-borne diseases can also be conveyed by furniture and other articles in common use. The portal by which most of these diseases enter the system is generally believed to be the lungs, but certain facts lately observed point to the tonsils, throat, and nose as possible channels for their introduction. Some of this group may be conveyed by milk, and it is possible that other ingesta may become contaminated by the contagia of these diseases. The air-borne diseases are as follows : Varicella, Scarlet Fever, Small-pox, Measles, Rubella, Diphtheria, Erysipelas, sometimes Influenza, Mumps, Rheumatic Fever, and Whooping-Cough. · Pulmonary Tuberculosis usually arises from the inhalation of contaminated particles.

(*b*) The *water-borne* group only comprises Enteric fever, Paratyphoid fever, Cholera, Dysentery. Two facts form the basis of the propagation and prevention of these diseases : (1) All matters coming from the patient's bowels and stomach are infective, in enteric the urine also ; and (2) to produce the disease the virus must be introduced by the mouth into the alimentary canal.

(*c*) The *third group* comprises disorders the infection of which is introduced into the blood or tissues of the body by means of a wound or a scratch which may perhaps have escaped notice, or by the bite of an insect. Our profession pays a penalty every year to this group of disorders when, perhaps, some overworked practitioner is called to the bedside of a *syphilitic* lying-in woman, and forgets to examine the margins of his finger-nails, where some crack or unsuspected scratch will be the means of the introduction of the syphilitic poison. Some of these disorders were formerly described as *miasmatic*—i.e., dependent upon some meteorological, telluric, or climatic influence, which we did not understand. *Malaria* is an example of these diseases, but it is now known to be directly introduced into the blood of the patient by the bite of a mosquito. *Tetanus* is introduced through a wound or scratch which has become contaminated with the soil ; *plague* is conveyed by rat fleas. *Typhus* is introduced by the bites of lice. *Septicæmia* is due to the internal or external contamination of the blood-current, and all kinds of dust probably contain pyogenic—i.e., septicæmic—microbes. *Glanders* is contracted from horses by the contamination of a wound or scratch ; and *Anthrax* is contracted by woolsorters and others who come in contact with the hides of animals containing the contagion, and thus inoculate a scratch or inhale the dust. *Tuberculosis* is placed under this group because it is sometimes undoubtedly inoculated into a wound, giving rise to lupus vulgaris or verruca necrogenica on the hand. *Hydrophobia* must be inoculated, generally by the bite of an animal suffering from rabies. *Gonorrhœa* is conveyed either to the urethra or conjunctiva, but whether a breach of surface is necessary or not is not known.

It follows, therefore, that the **procedure for disinfection** differs somewhat in the case of air-borne diseases, water-borne diseases, and those introduced by the contamination of a wound or scratch.

1. For AIR-BORNE DISEASES—

(i.) The *linen*, before washing, should be left to soak in carbolic solution (1 in 80).

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[1] Some valuable data on this question were collected by the author from the Warrington Small-pox epidemic, 1901-1902.—Appendix to the Report of the Roy. Com. on Vaccination.

In any case, rather than leave clothes and linen exposed to the air, keep under water until they can be removed (a wineglass of carbolic acid to a gallon of water is roughly 1 in 80).

(ii.) *Clothes and Bedding.*—If a disinfecting oven (at a temperature of not less than 210° F. or more than 320° F.) or a steam-heated chamber at 212° F. is not available, they may be spread out in the room, and treated by sulphur (see below) or formalin spray. It is very doubtful if the fumigation of clothes by sulphur is of much use. *Washable articles should be plunged into a tub containing carbolic solution (1 in 80, vide supra, and then sent to the wash,* when they should be boiled.

(iii.) The *patient,* before returning to his friends, must have several warm baths, and be washed with carbolic soap. This is very necessary in diseases where desquamation occurs, and anointing with carbolised oil is recommended.

(iv.) *To Disinfect the Room.*—Close the windows and doors, and stop up all crevices. Melt some sulphur over a fire in a saucepan or small iron bucket, set it alight, and place it on an old tray in the middle of a room ; then shut up the room for twenty-four hours. Use 1¼ pounds of sulphur for every 1000 cubic feet—3 pounds for an ordinary sized room. The fumes are very suffocating, but they will not hurt anything if the air be dry, excepting brass, and this may be protected by smearing it over with vaseline. A whole house may be fumigated in this way from the basement by closing the windows, stopping up the chimneys with newspapers, and opening the doors of communication. Nowadays it is recognised that a gaseous is much less thorough than a fluid disinfectant, and the walls should be washed with perchloride of mercury or saturated with formalin sprays. Formalin may be used as a vapour in the same way as sulphur. It is conveniently supplied as candles, which may be burnt.

2. For WATER-BORNE DISEASES—

(i.) The *excreta,* if practicable, should be burned ; if not, before being removed they should be covered with chlorinated lime or carbolic solution (1 in 40, *vide supra*).

(ii.) The *underlinen,* towels, bedding, etc., must be boiled, or treated very carefully as in air-borne diseases.

(iii.) All *drinking-water* should be boiled if there is the slightest suspicion of its being contaminated by leakage, soakage (however small) from cesspools, drains, or the reckless casting of slops, and by flies.

(iv.) All food utensils must be disinfected ; dishes and water bottles must be protected from flies.

(v.) All handlers of food or every individual where large groups of men are crowded together as in armies, should be examined and treated if found to be " carriers," *i.e.,* apparently healthy persons in whose excreta the cysts of the amœba of dysentery, or enteric, paratyphoid or cholera germs abound.

List of common disinfectants : Extreme heat (200° F. or more, and preferably moist) ; fumes of burning sulphur (SO_2) ; chlorinated lime (1 lb. in 4 gallons water) ; chlorine, evolved from chlorinated lime by hydrochloric acid (spirits of salts) ; carbolic acid (a wineglass of carbolic acid to each gallon of boiling water is roughly 1 in 80 solution) ; formic aldehyde ; permanganate of potash (Condy's fluid) ; chinosol ; lysol ; sulphate of iron ; sulphate of copper ; creolin ; corrosive sublimate ; terebene ; thymol ; eucalyptol ; sanitas.

(3) Disinfection and the PREVENTION OF DISEASES INCLUDED IN OUR THIRD GROUP differs in each individual case. Thus septicæmia and tetanus almost ceased in surgical cases with the introduction of cleanliness and asepsis. Various tropical fevers are conveyed to man by the bites of mosquitoes, flies, fleas, and bugs. The prophylaxis of these conditions includes measures directed to the extermination of the insect responsible and avoidance of places in which they are known to be present. Thus in malarious districts mosquito nets and tents are necessary, and the pools or other stagnant water in which the larvæ of the Anopheles or other incriminated species live should be treated with kerosene. In places where plague is endemic the rats should be destroyed ; where bugs infected with disease are found it may be necessary to adopt such measures as burning the huts, etc., in which the eggs are likely to have

been deposited. Many of these insect pests cannot be satisfactorily dealt with by any means at present discovered, as full knowledge of their life-history is the necessary preliminary to effective steps for their destruction. Useful information on those points is supplied in the War Office Memoranda of Diseases of the Mediterranean area, 1917.

§ 421. **Diet in fevers** is a question of great importance. It should consists mainly of milk and meat juices. No more than 3½ pints of milk, usually less, per diem should be given, fresh—sterilised if necessary—or scalded (not boiled), in small quantities at a time ; and it may often with advantage be diluted with half or a third of water, soda-water, or barley-water. If curds are passed, the milk may be peptonised, or sodium citrate may be added in the proportion of gr. 2 (0·13) to the ounce (32) of milk. Lime-water may be used instead if diarrhœa be present. If milk is not well tolerated, whey or cream may be given, or the yolks of eggs or egg-flip. Beef-tea, chicken or mutton broth, about a pint in the twenty-four hours, should also be given, and may be supplemented by some of the many modern substitutes (*e.g.* Liebig's or Valentine's extract, Bovril, etc.). Where the intestinal canal is much affected meat extracts and jellies should not be given. Some methods of preparing invalid foods are given in § 236. Iced water is very agreeable, but it generally increases the thirst. Fresh lemonade may be advantageously substituted by mixing a drachm or two bi-tartrate of potash, with a little sugar, to the pint of water. Except in presence of abdominal distension, cold water *ad libitum* may be given with advantage.

§ 422. The **Treatment of pyrexia and hyperpyrexia** comprises six indications :

1. *Heat production can be diminished and heat loss increased* to some extent by means of *drugs*, known as antipyretics, such as antifebrine, antipyrine, and phenacetin. The first of these is most efficacious for reducing temperature, but it requires care, on account of its depressing effect on the heart, and the reaction which follows some hours later. Cryogenin gr. 10 to 15 (0·6–1) is the least depressing antipyretic. Quinine in full doses (say 5 grains (0·3) every three or four hours) may be given until the temperature comes down or physiological symptoms are produced (singing in the ears, deafness, headache, etc.). Salicylates, especially in rheumatic affections, and aconite are also useful. Among the more familiar but less efficacious febrifuges and diaphoretics are liquor ammoniæ acetatis, potassium nitrate, spiritus ætheris nitrosi, and camphor : also lemon drinks, dilute acids, and salines. Parthenine (an alkaloid derived from Partenum histerophus (Linnæus) has been known as a febrifuge for a long while to the country people of Havana, where its common name is Escoba Amarga. Febrifuge doses of 2 grammes may be given. It was tried with success in eighty patients by Dr. Ramirez Tovar.

2. *To aid the loss of heat* is a method of treatment called for in cases of hyperpyrexia (*i.e.*, when the temperature reaches above 104·5° F.), by means of the graduated bath, the wet pack, sponging, the application of ice-bags, or Leiter's Coil.

The Graduated Bath.—Place the patient in a bath one-third full of water at 90° or 95° F. Every five minutes reduce the temperature 5° until 60° F. is reached. If the patient's fever be not then reduced to 100° F. or lower, he may be left in a further quarter of an hour if his pulse be a fair strength. The pulse must be closely watched, and alcohol given if necessary.

The Wet Pack.—Take off the night-shirt and superfluous bedclothes, and place the patient on a blanket. Moderately wring a sheet out of ice-cold water and lay it along his side. Gently roll him over on to it, and completely envelop him in it, head and all, except the face, so that it is next his skin, without creases or air, between the legs and beneath the arms. Cover these latter with wet towels. Then put two cradles over the patient, and blankets over all. Leave him thus packed for twenty to forty minutes, until his temperature, taken in the mouth, is reduced to the required extent.

Tepid Sponging.—Lay the patient in a blanket and sponge him gradually all over with tepid water (about 75°). Do half the body at a time, the other half being covered up. Continue the process for twenty to forty minutes, until the fever is reduced.

The application of ice in large ice-bags for the head, chest, and abdomen has been

used when other means are not available, but the weight of the bags and their localised application are objections to their use. *Leiter's Coil* consists of a specially made coil of metal or rubber tubing through which cold water is continually running. This coil may be applied to the head, abdomen, or chest. Neither of these two last methods are recommended for fever cases.

3. To *diminish the work done by the internal organs* is another means of combating pyrexia. This may be done by diet (*vide supra*), and by promoting the action of the skin and bowels, in order to relieve the kidneys. Saline purges fulfil the latter indication (F. 46, 51, 55, and 63, are useful).

4. In all fevers it is necessary *to watch the heart* very carefully, and, if necessary, to steady it by means of strychnine and digitalis, or to aid its flagging powers by means of stimulants. The pulse should be examined several times a day in all fever cases, if only for this purpose. Injections of camphor form an excellent stimulant.

5. *Symptomatic treatment* may also be necessary, but this has been dealt with in the preceding pages. The constipation must be relieved by calomel or saline purges, the thirst by lemon water in sips (not ice), and the headache by phenacetin.

6. The last indication is *to watch for and treat complications* as they arise. The chief of these are (i.) cardiac (*vide supra*), and (ii.) delirium and insomnia. If the delirium be of the *raving* kind, chloral and bromides should be given in full doses ; if, on the other hand, it be of the *muttering* or typhoid variety, stimulants and ammonia are indicated. Insomnia may be relieved by the same treatment, and alcohol may be useful in this respect. (iii.) Pulmonary complications, (iv.) suppression or retention of urine, and (v.) collapse, are all dealt with elsewhere.

· CHAPTER XVI
GENERAL DEBILITY, PALLOR, EMACIATION

A FEELING of general weakness and lassitude is a symptom common to a great many diseases, but we are now concerned with those in which this is the only obvious, or at least the most prominent, symptom for which the patient seeks relief. Diseases in which debility is the chief symptom may be classified clinically into two great groups according to whether they come on acutely and are attended by pyrexia or not. Debility coming on acutely and attended by pyrexia was fully dealt with in the preceding chapter. There still remains a large group of diseases in which the weakness is of gradual onset, runs a chronic and indefinite course, and is unattended for the most part by any notable elevation of temperature ; and these diseases may be attended by pallor or by emaciation. Here we shall often meet with the beginnings of disease, beginnings which may, however, lead to a serious and fatal issue. It is, therefore, of the highest importance that an exact diagnosis should be made, and treatment adopted as early as possible.

The debilitating conditions mentioned in this chapter may be un-attended by any other symptom, or only by the pallor of anæmia or the wasting of malnutrition, and many give rise to no characteristic anatomical changes after death. Their pathology in some instances is extremely obscure, and its elucidation in the future must largely depend upon the co-operation of the analytical chemist with the physiologist and the physician, a large proportion of them being undoubtedly due either to some kind of autotoxic, hæmolytic, hæmogenetic, or other blood changes on the one hand, or to malnutrition and a profound disturbance of metabolism on the other.

PART A. SYMPTOMATOLOGY

§ 423. General Debility.—Malaise, lassitude, inability to complete a day's work, are some of the terms used to describe the symptom under consideration, which is essentially chronic in its course. The weakness is generalised, and it may affect the mind as well as the body, for there is not only a disinclination to take muscular exercise, but an inability to concentrate the attention or accomplish mental work. The weakness may vary in kind and degree from very slight malaise to a total incapacity

to move. Many diseases in this category are apt to be overlooked in their earlier and more curable phases. The patient may attribute his ailment to " slight digestive derangement," or think he has " been working too hard," or " wants a change," and perhaps he calls on his doctor " as he was passing " just to confirm his own diagnosis and " give him a tonic." These cases may tax the young practitioner's skill and tact in several ways. Fresh from studying instances of marked diseases in hospitals, he may regard these cases as trivial and " uninteresting " ; and even if he detects the beginning of some insidious malady the patient may meet his suggestion of serious ailment not only with surprise, but even with resentment and distrust. • Some tact, thérefore, is required, and the practitioner may find it wise to place himself in communication with some discreet friend or relative of the patient.

Fallacies.- The distinction of general debility from *paralysis* is not usually difficult, though patients with multiple peripheral neuritis, early paraplegia, general paralysis of the insane, bulbar paralysis, and various other forms of paresis, often come to my clinique complaining simply of weakness. Cases of *malingering* offer far greater difficulty in diagnosis from general debility, for in both cases we are almost entirely dependent upon the patient's own statements. The question of motive should be considered and an exhaustive examination made by the most up-to-date scientific apparatus, but even then we may in justice be compelled to give the patient the benefit of the doubt. My experience at the Paddington Infirmary taught me in many cases that it is only by keeping the patient under daily observation, and with the aid of intelligent, experienced, and well-trained nurses, that a correct conclusion can be gained. I have no doubt that large numbers of able-bodied malingerers gain admission to the infirmaries in Great Britain as cases of general debility. *Hysteria* and *neurasthenia* may require to be distinguished from debility. The *Causes* of debility are discussed in §§ 431 and 448.

Pallor of the Skin—*i.e.*, deficiency of its normal colour—is a frequent accompaniment of cases in which debility is complained of by the patient, and the experienced observer can detect considerable variations in the different varieties of pallor belonging to several diseases which will be alluded to shortly (§ 431 *et seq.*).

Fallacies.--Slight *jaundice* may resemble some forms of pallor. In *town-dwellers* who suffer from a deficiency both of fresh air and sunlight, pallor of the face is common. In certain " delicate " families a pale face is more or less normal. Europeans who have lived long in the *tropics* are habitually pale and " anæmic " looking, but the blood may not reveal any changes of anæmia. On the other hand, patients may occasionally present flushing of the face and redness of the lips, though undoubtedly suffering from anæmia. In many *nervous* conditions transient constriction of the vessels may cause a pallor which may be mistaken for anæmia. Many patients who " go white " with nervous emotion are mistakenly supposed to be anæmic ; in anæmia the pallor has always a waxy or yellow

tinge, which is absent in pallor of vasomotor origin. With a glance at the colour of the lips one can usually distinguish the conditions.

Emaciation, or loss of flesh, may also be associated with general debility, and its presence adds considerably to the gravity of a case, for it indicates either serious organic disease such as cancer or tubercle, or definite defect in the alimentation or metabolism of the body, such as is produced by intestinal trouble or chronic Bright's disease. It is manifested to the patient by his clothes becoming looser, or his face becoming thinner, and to the physician by pinching up a fold of skin between the finger and thumb. But the only reliable test is a definite loss of weight, and it is advisable at the outset to ascertain and record the weight of all patients who come to us complaining of debility. To ascertain the net weight, one-twentieth of the gross weight may be deducted for summer, and about one-eighteenth for winter, clothes. Every consulting-room should be provided with scales. The causes of emaciation are discussed in § 445 et seq.

Fallacies.— A normal loss of adipose tissue may occur about the climacteric, but the reverse is quite as usual. In advancing years loss of flesh, or the reverse, is normal in some families ; both are largely a question of heredity. Amyotrophy, unless generalised, is not apt to be confused with emaciation ; it is usually localised. The diet a person has been taking will, within certain limits, influence his weight considerably, and one who has been taking only nitrogenous food (*e.g.*, the so-called Salisbury diet) may be many pounds under his normal weight.

PART B. PHYSICAL EXAMINATION

§ 424. The physical examination of cases of general debility, pallor, or emaciation, comprise (1) EXAMINATION OF THE VISCERA ; (2) OBSERVATIONS ON THE WEIGHT, and in some cases on the TEMPERATURE ; and (3) AN EXAMINATION OF THE BLOOD.

1. An examination of the VISCERA should be very systematically and thoroughly conducted (see Scheme, pp. 6 and 7), because we may be dealing with some incipient disease, the signs of which are obscure. Inquiries should be specially directed to the state of the digestive organs, and the urine should be carefully examined. Special importance will attach to the latter when we know more about the causes and consequences of abnormal hæmolysis (blood destruction).

2. The WEIGHT of the patient should be noted, and, if possible, compared with previous records. It may be desirable also to take the patient's TEMPERATURE if any pyrexia be suspected, and to obtain a series of records (§ 374).

3. An examination of the BLOOD is necessary, especially in cases where any form of anæmia is suspected. This in its complete form consists of (1) estimation of hæmoglobin ; (2) blood-counts of the red and white corpuscles ; (3) examination of blood-films. In most cases these three will be sufficient for a routine examination ; but in other cases it is

necessary to make (4) an examination for parasites and other abnormal constituents ; and (5) certain physical and chemical properties of the blood.

EXAMINATION OF THE BLOOD

§ 425. **Apparatus and Methods.**—APPARATUS REQUIRED.—A Tallqvist hæmoglobin scale or Sahli's hæmoglobinometer ; a Thoma-Hawksley hæmocytometer ; a sharp needle (the triangular surgical needles are very useful) ; a bottle of Hayem's solution ; a bottle of Toison's fluid ; a bottle of Geimsa or Leishman's stain ; a bottle of distilled water ; a bottle of alcohol ; a bottle of ether ; some squares of butter-muslin for cleansing lenses, etc. ; white filter-paper (blotting-paper) ; a case for holding slides ; slides and cover-slips ; a pair of rubber bellows for drying pipettes ; and a bulb and stem for cleaning pipettes. Cover-glasses and slides must be perfectly clean and free from greasiness. Cover-glasses should never be laid flat on the table, but have one edge on the table, the other edge leaning on some object.

For the *estimation of hæmoglobin* Sahli's hæmoglobinometer is the one most used. For *counting the blood-cells* the Thoma-Hawksley hæmocytometer is employed. Diluting solutions are required for this purpose. For counting the red cells a solution of normal saline may be used, or Hayem's solution ; sod. chlqride 1 grm., sod. sulphate 5 grms., hydrarg. perchlor. 0·5 grm., aq. dest., ad 200 c.c. For counting the white cells a 0·3 per cent. solution of acetic acid coloured by methylene blue is used, or Toison's fluid (methyl violet, 0·025 grm. ; neutral glycerine, 30·0 c.c. ; distilled water, 80·0 c.c. Add to this a solution of sodium chloride 1·0 grm., sodium sulphate 8·0 grm., distilled water 80·0 c.c., and filter). The instruments must be carefully cleaned before being put away, first with water, then with alcohol and with ether, and then dried.

METHOD OF OBTAINING BLOOD.—Certain precautions are necessary to obtain satisfactory results in procuring a specimen of blood for examination. Any series of observations on the same patient should be carried out as far as possible always under the same conditions, because there are physiological alterations in the constituents of the blood after meals, cold baths, exercise, etc. The necessary blood is to be obtained by puncturing the lobe of the ear with a surgical needle, a lancet such as is supplied with most hæmoglobinometers, or a steel pen of which one half the point has been broken off. The ear should not be first cleaned, as this is unnecessary, and alters the composition of the blood locally. The puncturing instrument must, however, be sterilised by heat or by keeping it in a small tube of alcohol. A sufficiently deep puncture should be made to obviate the necessity of squeezing the ear to procure enough blood. If it is only desired to take blood for the purpose of serum reactions, squeezing the ear is of no moment, but if for study of the cells it must be avoided. The practice of obtaining blood from the finger is unpleasant for the patient and inaccurate.

§ 426. **Estimation of the Hæmoglobin.**—In estimating the quality of the blood the number of red cells is of less importance than the amount of hæmoglobin, their active constituent. Hæmoglobin may be roughly estimated by the *Tallqvist scale*,[1] which consists of a lithographed scale of tints. A drop of blood is sucked up by one of the pieces of blotting-paper supplied, and compared with the scale of tints as soon as the stain has lost its humid gloss. The figures beside the tints represent the percentage of hæmoglobin present, normal being 100. The estimation can only be performed in full daylight.

Another method of estimating the hæmoglobin is by means of *Sahli's hæmometer* (see Fig. 108). The graduated tube is filled to the mark 10 with diluted (5 per cent.) hydrochloric acid. From the drop of blood a quantity of 20 c.mm. is sucked into the capillary pipette, and this blood is blown into the graduated tube containing the 1·5 per cent. hydrochloric acid. The pipette must be entirely emptied of its contents by alternate sucking in and blowing out, because the standard solution is regulated

[1] The Tallqvist scale can be obtained from Messrs. Allen & Hanbury, Wigmore Street, London, the authorised agents for England.

according to this scale. This alternate sucking in and blowing out is also the best means for thoroughly mixing the blood with the hydrochloric acid. In a few moments the solution will have acquired a dark brownish-yellow colour. Then the water is slowly added until the solution, when placed against the standard solution and examined in the light, exactly corresponds to the standard unit. The figure to which it has been necessary to fill the tube with water marks the percentage of the hæmoglobin of the blood, its normal condition having been fixed at 100. During the examination it is advisable from time to time to turn the graduated tube so as to bring its scale under the edge of the stand. This will prevent the operator from being influenced by the reading of the scale or a preconceived notion with regard to the possible amount of the hæmoglobin present. To guard against the latter eventuality, it is useful to screen the entire upper part of the tube with a piece of opaque paper, so that one cannot see how high the fluid has risen in it. As we observed before, the
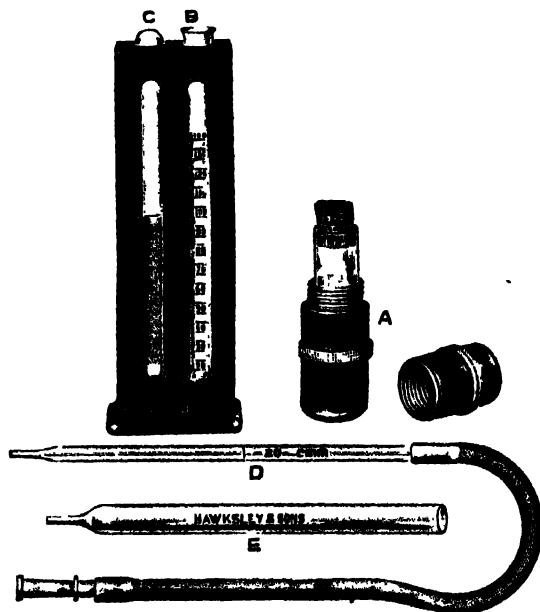
FIG. 108.— SAHLI'S HÆMOMETER.

examination can take place under any light and, contrary to other methods of experimenting, gives the same results whether done by natural or artificial illumination.

Significance of Diminution or Increase of Hæmoglobin.—The amount of hæmoglobin is always expressed in terms of a percentage of the normal standard. Thus, 87 on the scale indicates that the amount is 87 as compared with the normal of 100. A diminution of hæmoglobin is the essential feature of all anæmias, but the various forms of anæmia differ as regards the number of blood-cells. The hæmoglobin may be rapidly estimated in the consulting-room by the Tallqvist scale ; it is useful, for example, to gauge the degree of progress a patient is making under treatment for anæmia. In chlorosis there is a marked diminution of hæmoglobin in each corpuscle, though their number may not be much diminished. On the other hand, in pernicious anæmia the diminution of hæmoglobin is due to the diminution in the number of red cells, each of which contains the normal or above the normal hæmoglobin value. The amount of hæmoglobin in each corpuscle is expressed by making a fraction, the numerator of which is the percentage of hæmoglobin present (as estimated by the

hæmoglobinometer) and the denominator of which is the percentage of corpuscles, taking 5,000,000 as normal. For example, if the examined blood has 40 per cent. hæmoglobin and 80 per cent. red corpuscles, the value of hæmoglobin in each corpuscle is $\frac{40}{80}$ths normal. This fraction expresses the *colour index* of the blood, or ratio between the percentage of hæmoglobin and the percentage of red corpuscles. If the colour index is much below unity, the anæmia present is of a chlorotic type, and in most cases the prognosis is good. On the other hand, if the colour index is 1·2 or higher the probability that pernicious anæmia is present is strengthened. In dealing with cases of cyanosis the amount of hæmoglobin may enable the physician to decide whether the case is one of true polycythæmia or one dependent upon drug-taking. In the latter the hæmoglobin is rarely increased; in true polycythæmia it usually is so. In town-dwellers the hæmoglobin is usually only 80 to 90 per cent. ; in out-of-door workers it may be over 100 per cent.

§ 427. Blood Counts.—An estimation of the number of corpuscles in the blood is in many cases of extreme importance both for the diagnosis and the treatment of disease. We recommend the method of estimation of the NUMBER OF RED CELLS in a cubic millimetre of blood, by means of the *Thoma-Hawksley hæmocytometer.* The apparatus consists of a mixing pipette, a graduated counting slide, and a diluting fluid (§ 425). Before starting see that all the instruments are clean and at
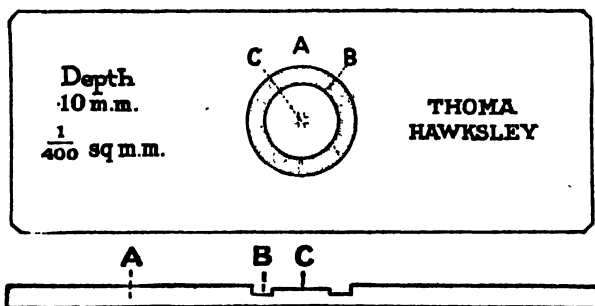

FIG. 109.—THE THOMA-HAWKSLEY HÆMOCYTOMETER.

hand. Suck up the blood from the drop to mark 0·5 into the capillary pipette; if any of the blood reach the mixing chamber the instrument must be cleansed, and the process started again, as it is necessary to be very precise in this measurement. Wipe rapidly the end of the pipette, plunge it into the diluting fluid, and suck it up to the mark 101. To ENUMERATE the LEUCOCYTES a special pipette, marked 11 above the bulb, is supplied. It gives a dilution of 1 in 20 if the blood is sucked up to mark 0·5. The diluting fluid is drawn up to the mark 11. Holding the pipette between the finger and thumb, rotate and shake it so as to thoroughly mix the fluids in the mixing chamber; the glass ball greatly facilitates this process. [If the blood is collected at the bedside, it is necessary to carry it elsewhere before the counting can be done. To ensure its safe conveyance, remove the mouthpiece from the rubber on the end of the pipette, and turn the free end of the rubber over the other end of the pipette. They must be carried horizontally, or the result will be valueless.] Next blow out and discard the clear fluid from the capillary end of the tube, and also three or four drops of diluted blood; then let a fraction of a drop fall upon the graduated platform in the centre of the slide. The drop must be of such a size that when the cover-glass is applied the blood will not run over the edge into the trench around. Place the cover-glass on the drop of blood, and if the cover-glass has been properly cleansed it lies so closely on the outer rim that Newton's concentric colour rings can be observed on the cover-glass. Set the slide for a few minutes to allow the corpuscles to settle. The platform is ruled by cross lines each enclosing a space of $\frac{1}{400}$ of a

square millimetre ; the depth of each square with the cover-glass on is $\frac{1}{10}$ of a milli-metre. The squares are marked out into sets of 16 by double lines. Count the red cells in five of such sets—that is, in 80 squares. Where the corpuscles lie *upon* the lines count those on the upper and left side lines only. *Calculation for counting red blood-cells* in 1 c.mm. of blood : The 400 squares equal $\frac{1}{10}$ c.mm. ; 80 squares are counted, and equal $\frac{1}{50}$ c.mm. They are found to contain, for example, 480 corpuscles. Adding 0000 to the number counted gives the number per c.mm. For $\frac{1}{50}$ c.mm. contains 480 cells ; therefore 1 c.mm. contains 480 × 50 × 200—*i.e.*, 480 × 10,000 $=$ 4,800,000 cells.

Calculation for counting white blood corpuscles or leucocytes : The 400 squares are counted, and contain, say, 43 leucocytes. The 400 squares are counted again, and contain, say, 37 leucocytes. Now, since 400 squares equal $\frac{1}{10}$ c.mm., 800 squares equal $\frac{1}{5}$ c.mm., therefore $\frac{1}{5}$ c.mm. contains 43 + 37 $=$ 80 leucocytes, and 1 c.mm. con-tains 80 × 5. But the blood is diluted 20 times, so that 1 c.mm. of blood contains 80 × 5 × 20—*i.e.*, 80 × 100 = 8,000 leucocytes (*i.e.*, add 00 to the number counted).

Significance of Diminution or Increase of Red Cells.—In health the average number of red cells per cmm. is about 5,000,000 in the male and 4,500,000 in the female. It is increased to 7,000,000 or 8,000,000 in the newly-born, in plethoric persons, after fasting and sweating, and after removal to high altitudes. With menstruation, child-birth, and the drinking of much fluid there is a decrease. In *disease* there is an increase per c.mm. (i.) in cases in which there is defective oxygenation in the lungs, as in chronic lung disease or other cause of obstruction to the free entry of air ; (ii.) in cases where the blood becomes concentrated owing to loss of fluid, as after diarrhœa, vomiting, polyuria, rapid pleural or other effusions; and (iii.) in cases where the blood passes too slowly through the lungs owing to cardiac insufficiency or bradycardia. In con-genital heart disease the number may be 10,000,000 or more per c.mm. It is also met with after severe burns, after the use of iron, in phosphorus poisoning, and in the over-production following hæmorrhage, and in erythræmia (§ 29). It does not occur with cyanosis *per se*, but only when cyanosis is accompanied by one of the conditions above mentioned. Diminution in number is found after hæmorrhage and other secondary anæmias, and in leukæmia. In pernicious anæmia the diminution may be very great, and in chlorosis very slight.

Significance of Increase or Diminution of the Leucocytes.—In health the normal number of leucocytes is 6000 to 10000 per c.mm. On a normal field, with $\frac{1}{6}$ English objective, and a No. 2 eyepiece, about three or four white cells are generally seen. In the newly-born there may be over 17,000 and up to seven years of age from 10,000 to 14,000 leucocytes per cmm. During pregnancy, after meals, cold baths and exercise, there is an increase in the number of leucocytes. The polynuclear neutrophil leucocytosis (§ 428) is the most usual form of leucocytosis both in health and disease. In some fevers the leucocytes are increased (leucocytosis), in others diminished (leucopenia). This may be of great diagnostic importance, especially in cases in the tropics ; such variations are described under each fever. Generally speaking, any collection of pus will give rise to a leucocytosis ; if free exit be provided, the leucocytosis falls markedly or disappears within thirty-six hours. If the exit has not really drained the whole cavity, the leucocytosis does not fall, and this may afford an indication for further operation. The amount of the leucocytosis is not pro-portionate to the amount of the pus. Leucocytosis may be absent when the pus is well walled off, as in chronic abscesses, and also when the patient is not reacting to the toxin. In the latter case the prognosis is uniformly bad. Leucocytosis is caused by certain drugs, by convulsions of any sort, and by heat stroke. Cancer, except in the case of very small growths, commonly causes a leucocytosis ; if after the removal of a primary growth leucocytosis is found to persist or to recur after having disappeared, the presence of metastases is extremely probable. The following diseases may be mentioned as among those in which the presence of leucocytosis is likely to be of diagnostic importance : Abscess and Suppuration, Septicæmia, Pneumonia, Erysipe-las, Scarlet Fever, Osteomyelitis, Malignant Endocarditis, Tuberculous Meningitis, Cancer, and Pertussis.

Various blood diseases give rise to leucocytosis, and in these it is of importance to study particularly which type of leucocyte is increased ; for this purpose *staining of blood-films* is necessary.

§ 428. Microscopic Examination of Blood and Blood Films.—Alterations in the shape and size of the blood-cells may be seen by examination of fresh blood-films, but for accurate examination of the structure of the red and white cells and a differential count of the leucocytes it is essential that blood-films be fixed and stained. Blood may be obtained from the lobe of the ear by the method above described (§ 425). A microscopic examination of fresh blood may be made by applying a clean slide lightly to the drop of blood, placing a cover-glass on it, and examining under the microscope. For this method any good microscope will do with a ⅙ or a ¼-inch English objective (or a Zeiss's D) and a No. 2 eyepiece ; but for the differential examination of leucocytes and for bacteria a ¹⁄₁₂-inch oil immersion lens is necessary. It is a great advantage to have a nose-piece on the microscope capable of carrying two or three objectives, so that one can first examine the specimen with a low, and then with a high power. It is well to make oneself familiar with the changes the blood undergoes in a short time after such a method of preparation. If it be desired to preserve such a specimen for some hours, ring the edge of the cover-glass with vaseline to prevent the entrance of air. In this simple way we are able to note any abnormality in the shape of the red cells, or the presence of abnormal constituents, such as particles of pigment, filaria sanguinis hominis, or the spirillum of relapsing fever. We may also note any excess of white cells. Rouleaux formation is also noted in normal fresh blood—*i.e.*, the red cells run together, leaving clear the concave spaces in which blood platelets are seen. The white corpuscles are spherical, clear, and nucleated.

A film may be made upon a cover-glass or a slide ; for ordinary purposes the slide method is the easier. It is essential that the slides or cover-glass be absolutely clean and free from greasiness. Lay the surface of the slide lightly on the drop of blood exuding from the lobe of the ear, and with the smooth edge of another slide spread out the blood in a *thin* film by pushing the drop along the surface, so that it forms an even film. Care must be taken not to handle the slides too much or to breathe upon them. Allow the film to dry, and it will if necessary keep for several days without further precautions.

Staining.—The protoplasm of the red cells take up acid dyes only ; normal nuclei take up basic stains. The granules met with in the protoplasm of the various leuco-cytes take up different stains ; some have an affinity for acid stains such as eosin, and are known as oxyphil or eosinophil granules ; some take up basic stains such as methylene blue, and are called basophil granules. The granules occurring in the ordinary polynuclear leucocyte were at one time supposed to take up both acid and basic granules, and hence were named neutrophil granules. It is now known that these granules take up faintly acid stains, though the cell is still named polynuclear neutrophil for purposes of description and differentiation. In staining we therefore employ a triple stain which allows each part of the blood cells to take up the dye for which it has affinity. When the film is dry, place it in methyl alcohol for 3 minutes. Allow it to dry. Place it on a horizontal surface and cover with equal parts of distilled water and Leishman's or Geimsa's stain previously mixed in a test tube. After three minutes pour off, wash rapidly in distilled water, and allow to dry. Drying with blotting-paper tends to spoil films. By further diluting the stain and allowing it to act for a longer period better results may be obtained.

Variations of the Red Blood Corpuscles in Disease may consist of (1) variability in form (poikilocytosis) ; (2) variability in size ; (3) nucleation ; (4) polychromatophilia ; and (5) stippling. Normal red cells are circular, bi-concave, non-nucleated discs measuring in size 6 to 8 μ or $\frac{1}{3200}$ of an inch.

Poikilocy.   (Fig. 110) is a variability in the shape of the red cells. They may resemble a flask, a pear, or a kidney. This change used to be regarded as pathog-nomonic of that serious disease pernicious anæmia ; but the change is also found in leukæmia, splenic anæmia of children, and in severe secondary anæmia such as
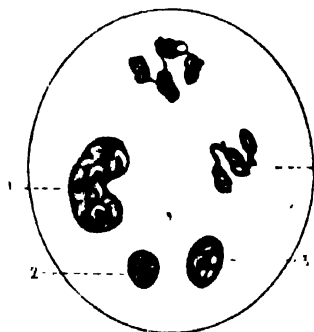
PLATE III.

FIG. 1. FIGURE TO SHOW VARIETIES
OF NORMAL LEUCOCYTES.

(*Obj. oil immers. $\frac{1}{12}$ —Eyepiece 3*)

1, Large mononuclear leucocyte; 2 and 3,
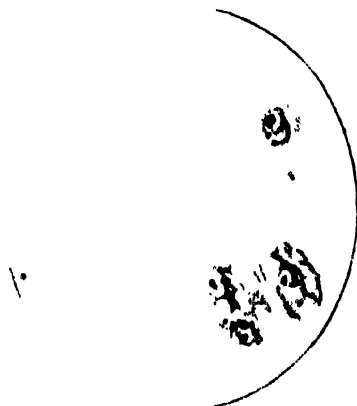lymphocytes; 4, polynuclear leucocytes

FIG. 2.—STAGES OF THE MALARIA
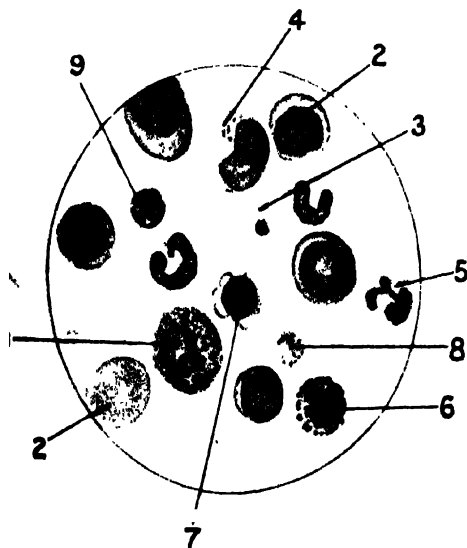PARASITE IN THE RED BLOOD
CELLS.

FIG. 3.—SPLENO-MEDULLARY
LEUKÆMIA.

(*Obj. oil immers. $\frac{1}{12}$ Eyepiece 3. Leish-
man's Stain.*)

1, Eosinophilic myelocyte (coarse granules),
2, myelocyte, 3, nucleated red corpuscle 4, mye-
locyte with fine eosinophil (oxyphil) granules,
intermediate between ordinary myelocytes and
eosinophilic myelocytes, 5, polynuclear leuco-
cyte, 6, mast cell, 7, megaloblast, 8, stippled
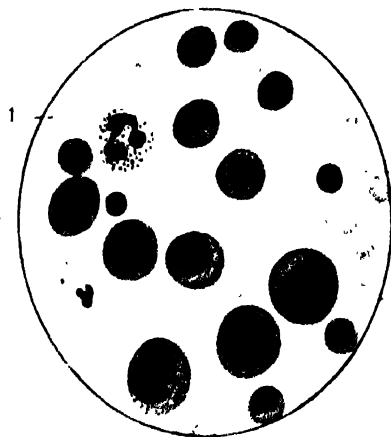red corpuscles, 9, lymphocyte

FIG. 4.—LYMPHOCYTHÆMIA (LYM-
PHATIC LEUKÆMIA).

(*Stained with Leishman's Stain.*) ×500.

1, Polynuclear leucocyte, 2, small, and
3, large, lymphocyte-like cells

occurs in cancer, nephritis, and malaria. With variation in shape the red cells in any form of profound anæmia appear to undergo degenerative changes—clear hyaline spaces (vacuolation) are seen inside the corpuscles when the specimen is examined just after removal from the body. It must be remembered, however, that such spaces as these may also be seen in normal blood about an hour after it leaves the body. Other more definite changes are seen in the staining of the cells, for whereas normal red cells take up only acid stains, these cells take up both acid and basic stains, and their substance stains irregularly. This property is known as *polychromatophilia*. Basophilia or " *stippling* " is another abnormal staining reaction of the red cells. With a mixed dye they appear to contain minute dots staining blue. Both polychromatophilia and stippling are seen in anæmic blood ; the former is almost certainly due to the youth of the cells and indicates not degeneration, as was formerly taught, but an exceptional call on the marrow. The significance of " stippling " is not so certain ; it is one of the earliest symptoms in lead poisoning, and may follow the ingestion of other metals also.

Variability in Size.—The normal red corpuscle measures about 7 μ ; red cells measuring under 6 - μ are termed microcytes, and those measuring over 8 μ, megalocytes. Both variations are met with in pernicious anæmia, leukæmia, the splenic anæmia of children, and in severe secondary anæmia. They are also seen in severe cases of chlorosis.

Nucleated red cells are found in all cases where there is a great diminution in the number of the red corpuscles—as, for example, in pernicious anæmia, severe secondary anæmia ; and in spleno-medullary leukæmia even without much diminution of the red cells. They are rare in chlorosis. These nucleated red cells must be distinguished from lymphocytes, which resemble them approximately in size. The
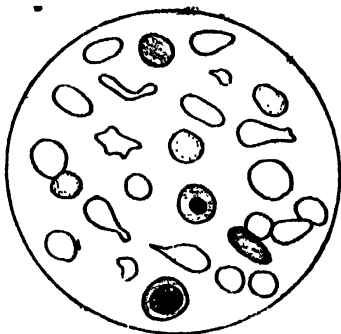

Fig. 110.—Diagram by Dr. Gordon Ward. Blood in pernicious anæmia ; showing poikilocytosis, polychromatophilia, basophil stippling, a normoblast, and a megaloblast.

nucleated red cells differ in the more homogeneous staining of the protoplasm. The nuclei may show karyokinesis, or may be degenerate. There are three distinct forms of nucleated red corpuscles : the normoblast, about the same size as a normal red cell; the large form or megaloblast, which is about three times the size of an ordinary red cell ; and the microblast, which is smaller than the normal red cell. The presence of the megaloblast in the blood is generally of grave import, indicating the presence of rapid regenerative changes in the blood. These cells are usually polychromatophilic (*vide supra*). They may occur in large numbers in pernicious anæmia.

Variations in the Leucocytes (Plate III., Fig. 1) may occur in regard to their absolute number (blood-count, *vide supra*), their structure, and the relative number of one kind or another (differential count). There are several kinds of leucocytes, and it is possible to identify the cause of an increase in the leucocytes by the predominating variety present. For this purpose and in order to make a differential count to ascertain the relative proportion of the several varieties, it is necessary to employ the staining method given above.

The varieties of leucocytes found *in health* are as follows :
In 100 leucocytes :

Polynuclear	60 to 70
Small mononuclear leucocytes (lymphocytes)	20 to 30
Large mononuclear leucocytes	2 to 5
Transitional forms	2 to 5
Eosinophil cells	1 to 3
Basophil or mast cells	0·5 to 1

(1) In the *Polynuclear Neutrophil Leucocytes*, which form 60 to 75 per cent. of all leucocytes in the blood, and have an average diameter of 13·5 μ, the nucleus is long and lobed, giving the appearance of being multipartite, and the protoplasm of the cell contains fine neutrophil granules. The so-called neutrophil granules are really faintly acid. This cell originates from the bone-marrow, and is actively amœboid and phagocytic (microbe devouring). (2) *Lymphocytes*, 20 to 30 per cent., are small cells without granules, with one large nucleus and very small amount of surrounding protoplasm : coming chiefly from the lymphatic glands and adenoid tissue. They are neither amœboid nor phagocytic. (3) *Large mononuclear* or " hyaline " cells have one large spherical nucleus and a larger amount of protoplasm than the lymphocytes. These cells are supposed to come from the bone-marrow, and are slightly amœboid. (4) *Eosinophil polynuclear* leucocytes, with coarse, eosin-staining granules, coming from the bone-marrow, are amœboid, but not phagocytic. (5) *Basophil* leucocytes, or mast cells, with coarse basophil granules. (6) *Transitional forms*. These are of uncertain origin. Their nuclei are horseshoe shaped and stain less heavily than those of polynuclear cells.

Variations of Leucocytes in Disease.—Variations in the number of the leucocytes in health have been referred to (p. 593), and a number of causes of simple increase in number of leucocytes has been given. Polynuclear neutrophil leucocytosis is the commonest form of increase of the white cells. When leucocytosis is present, a differential count is desirable. At least 500 leucocytes must be counted, the number of each variety noted on a piece of paper, and the percentage of each calculated. In many cases it may be necessary to make this count daily—*e.g.*, to watch the steady daily increase of polynuclear neutrophil cells, which may denote a peri-hepatic or other obscure abscess within the body. Except in leukæmia, leucocytosis rarely passes beyond 100,000 per c.mm. (normal about 7000 per c.mm.).

(1) The *Polynuclear Neutrophil* cells constitute, as just mentioned, the majority of the white cells in the blood. They are greatly increased in infective diseases, such as pneumonia, septicæmia, erysipelas, cerebro-spinal meningitis, scarlet fever, and in local inflammations or abscesses such as appendicitis and osteomyelitis. So true is this that an unfavourable prognosis in croupous pneumonia can be based upon an absence of leucocytosis. In typhoid fever the onset of a suppurative complication may be diagnosed by the presence of an increased number of polynuclear neutrophil leucocytes. A diagnosis may be made between typhoid fever and tuberculous meningitis from the fact that in meningitis there is leucocytosis, but there is none in typhoid fever uncomplicated by abscesses. If leucocytosis be found, even without definite physical signs pointing to an inflammatory condition, the onset of inflammation can almost with certainty be predicted. In this way blood examination comes to be of the highest importance in the diagnosis of obscure abdominal cases, as a polynuclear increase is rightly regarded in such cases as an indication for operation. The blood signs of deep-seated suppuration are increasing leucocytosis, with a high percentage of polynuclear leucocytes, increase of blood platelets, and glycogenic degeneration of white cells—*i.e.*, staining with iodine. In cancer of the stomach it has been found that there is often no increase of leucocytes one hour after meals, as would occur in health or in simple ulcer of the stomach. In all severe stomach trouble leucocytosis after digestion is diminished or absent. After profuse hæmorrhage and in malignant cachexia leucocytosis is present. The subcutaneous injection of irritants also produces leucocytosis.

(2) An *increase of eosinophil* cells occurs in one form of leukæmia (see below), and in several skin diseases, notably pemphigus and psoriasis. In asthma they may be increased to 25 per cent. or more, and this may be an aid to its diagnosis from cardiac disease and mediastinal tumour. Local accumulations of eosinophils also occur in the bronchial secretion of asthma and sometimes around cancer growths. Eosinophilia is found in those suffering from hydatid, trichinosis, and ankylostomiasis ; indeed, in obscure cases its presence may give the physician the clue to search for

parasites. It also occurs in chorea, Hodgkin's disease, after tuberculin injections, and is one of the salient features of anaphylaxis (see § 418).

(3) *Lymphocytosis* (increase of lymphocytes) occurs in lymphatic leukæmia (up to as high as 99 per cent.), whooping-cough, tuberculosis (except meningitis and sometimes acute miliary tuberculosis), glandular fever and after secondary syphilis. The diagnosis of Hodgkin's disease from lymphatic leukæmia may depend upon the examination of the blood. In the former there is no leucocytosis, or if present there is an increase mainly of the polynuclear leucocytes ; in the latter the lymphocytes are greatly increased.

(4) *Other forms of leucocytes* which make their appearance in disease are (i.) myelocytes, very large cells, mononuclear, some with fine neutrophil, some with coarse eosinophil granules, coming from the bone-marrow. Myelocytes occur in the splenomyelogenous form of leukæmia in large numbers (30 per cent.), and in anæmia splenica infantum, and in all diseases in which there is an excessive call upon the blood-forming activities of the bone-marrow. (ii.) Immature cells of various sizes and staining reactions may be seen in similar conditions.

Melanæmia is a term applied when certain pigment granules occur in the blood after malaria, relapsing fever and some melanotic tumours. They appear either in minute black lumps, or are enclosed within the cells.

Lipæmia.—The blood is laden with fat and of a pale pinkish colour in some cases of diabetes.

Iodophilia.—In suppurative and other disorders the plasma of the leucocytes has an affinity for iodine. Place the blood-films in a stoppered bottle containing crystals of iodine and leave for two hours ; then examine under a high-power lens. If iodophilia is present there are black dots or a diffuse dark coloration in the leucocytes. In the normal film the red cells are stained orange, the leucocytes are unstained. The reaction is said to depend upon the presence of glycogen in the plasma. By this method the presence of organic disease may be diagnosed in obscure cases.

Blood Platelets.—The blood platelets are seen with 1½-inch oil immersion lens as irregular bodies, small, apt to run together in clumps. They are concerned with coagulation and generally speaking are reduced in number in purpura. The normal number is about 210,000 per c.mm.

Blood Dust is a term which has been given to a number of small clear bodies in the blood, with a vibratile motion, about ¼ to 1 µ in size. They are supposed to be granules extruded from neutrophil and eosinophil cells.

§ 429. Parasites found in the Blood.—The MICRO-ORGANISMS which can be detected in the blood, and their methods of detection, as far as it is applicable to clinical work, are referred to in Chapter XX. The chief animal PARASITES which have been found in the blood are the microfilaria sanguinis hominis, Bilharzia hæmatobium, the malaria parasite, the protozoon of kala-azar, and the trypanosoma.

The PARASITE OF MALARIAL FEVER is a protozoon, inhabiting the red corpuscles, which it destroys, but it does not invade other tissues. There are three well-marked species of the parasite, distinguished from each other by their intracorpuscular development, and these varieties correspond to the three types of malaria known as benign tertian, quartan, and malignant tertian fever. The life-history of the protozoon runs through two stages : (i.) The asexual or intracorpuscular stage in man, and (ii.) the sexual form within the body of a mosquito belonging to the genus Anopheles (Fig. 98, § 408). The tertian parasite, which gives rise to the *benign* form of malaria, is the least virulent (Fig. 111). It is first seen within the corpuscle as a small, clear, ovoid body about 2 µ in diameter, possessing active amœboid movement. It gradually increases in size, and after the lapse of a few hours becomes ring-shaped, with very finely granulated pigment collecting about its centre. In the benign tertian form there is at the same time marked enlargement of the corpuscle, with a striking decrease in its colour. At the stage of full growth the parasite occupies nearly the whole of the enlarged corpuscle, and now it may follow either of two lines of development : (i.) The pigment gathers in the centre of the parasite, the chromatin divides and

the protoplasm is arranged around these masses so that rosettes of from fifteen to twenty segments are formed ; these are set free as spores by the rupture of the red blood corpuscles containing them, and masses of insoluble pigment enter the blood-stream. This phase is known as " segmentation " ; it is complete in about forty-eight hours, and corresponds clinically to a fresh paroxysm of the fever. The intracorpuscular development just described is asexual. (ii.) The sexual development takes place in the female mosquito, which sucks the blood of a malarial patient, and takes into its stomach the protozoon in the phases above mentioned. All die except the male and female gametocytes (which are crescentic in the malignant tertian type), the female being distinguished by the more compact arrangement of chromatin and pigment. The male gametocyte develops within the stomach of the mosquito into a flagellated body, and one of the flagella unites with the female gametocyte. The fertilised gametocyte (the travelling vermicule) penetrates the wall of the stomach and comes to
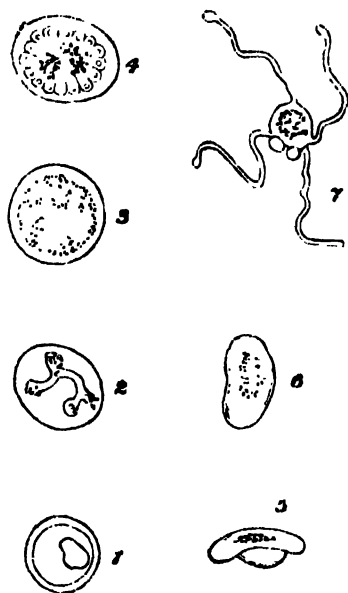
FIG. 111.—PARASITE OF MALARIAL FEVER.
1 to 4 = stages in benign tertian ; 5 to 7 = stages in malignant tertian ;
5 and 6 crescents ; 7 = flagellated body.

lie on its outer surface where it projects into the body cavity of the insect, and becoming spherical it develops within it (the sporocyst) a large number of curved, needle-shaped bodies. These are set free and reach the salivary gland of the mosquito from whence they are injected into the blood of the person bitten, and enter his red corpuscles.

The parasite of the *malignant tertian* fever is first seen in the red blood-cells as a tiny, unpigmented, hyaline body, forty-eight hours being needed for its development. At first it exhibits energetic amœboid movements, but ultimately settles into a bright, colourless, ring-like form, with one or two pigment granules contained therein. There is frequently multiple infection of a single corpuscle. The rosette or sporulating stage is rarely seen in the peripheral blood. In about a week (during the period of remission) characteristic crescent bodies, containing masses of coarse pigment granules, begin to appear, and increase in number rapidly. They are incapable of sporulation, and represent the sexual form—the gametocyte.

The *quartan* parasite, the easiest form for the beginner to study because of its

visibility, first appears as a small, round, clear speck, resembling a vacuole, but with feeble amœboid movement. It takes from sixty to seventy-two hours to complete its cycle. By the third day pigment, coarser and blacker than that of the tertian form, gathers round its periphery. On the fourth day segmentation takes place, the pigment flows in towards the centre, and here forms the radiating lines which produce the beautiful " daisy rosette " so characteristic of the quartan parasite. It breaks up eventually into eight to ten spores, and these with the insoluble pigment become free in the blood-stream. The development of the gametocyte resembles that of the benign tertian variety. There is no enlargement of the red corpuscle.

STAINING. Employ Leishman's method. The protoplasm of the parasites is stained bright blue, the chromatin pink, the granules are nearly black (Plate III.).

SCHISTOSOMA HÆMATOBIUM and allied parasites are described elsewhere (§ 326 and § 249), under the chief symptom they produce.

FILARIA BANCROFTI is a parasite which occasionally produces elephantiasis, chyluria, etc. The method of revealing the embryo is to allow a thick drop of blood spread upon a slide to dry. Stain half a minute in a 2 per cent. solution of methyl blue. Decolorise if necessary in dilute acetic acid (4 drops in 1 ounce of water), and examine with low power (see Fig. 112).

Filaria bancrofti rarely causes any pathological symptoms in the embryonic state. The embryos may be found in 20 per cent. of apparently healthy residents in Barbadoes and other tropical countries. The embryo comes into the peripheral blood at night (from 6 P.M. to 10 A.M.); the maximum number is usually found about midnight. It may be necessary to make repeated examinations at intervals of two hours to find them. During the day the microfilariæ go into the lung blood-vessels, where they are usually found in autopsies of people who had them, and died in the daytime. Should a victim of the parasite alter his usual habits, and sleep during the day, the filaria periodicity is reversed. The adult filariæ inhabit the lymphatics, where they give birth to immense numbers of embryos, a large number of which must in some unknown way disappear, or else the blood would contain them in incalculable numbers. Adult parasites after their death cause well-marked symptoms—viz., various forms of elephantiasis, lymph-scrotum, hæmatochyluria, chylous diarrhœa and ascites, usually related to their blocking of the lymphatic circulation.

FIG. 112.—FILARIA SANGUINIS HOMINIS.

TRYPANOSOMA.—The parasite of *trypanosomiasis* (§ 415) is a flagellated protozoon (Fig. 113). It is usually obtained in trypanosomiasis by gland puncture, and can also be found in the blood and cerebro-spinal fluid. It is found free in the blood. One end of the parasite is drawn out into a whip-like process, the flagellum; the other end is bluntly conical; the body itself is short and thick, and its substance granular. It contains a trophonucleus and a kinetonucleus. Attached to one side is a transparent, flange-like process, the undulating membrane. The length of the parasite, including the flagellum, is about 18 μ to 25 μ. It is best stained by Leishman's or Romanowsky's stain.

The protozoa of KALA-AZAR are found in the spleen, liver, bone-marrow, the blood, and in the lymphatic glands from the mesentery. They have been chiefly studied in blood and pulp withdrawn from the spleen, or preferably the liver during life. The commonest form found is a small ovoid body longer than it is broad, below 2 μ in diameter, measuring about one-fifth of a red corpuscle in its longest axis. It contains two nuclei; one is small, rod-shaped, and stains deeply; the other is larger, rounded,

and stains less deeply.   Other forms met with consist of small groups of similar bodies clumped together, resembling a quartan sporulating, malarial parasite. They stain faintly with methylene blue ; but the best method of staining them is by Leishman's or Romanowsky's stain.   Similar bodies have been found in Delhi boil and infantile splenomegaly.   They are known as Leishman Donovan bodies, and outside the body they elongate and develop a flagellum.

§ 430. Physical and Chemical Properties of the Blood.—The ALKALINITY OF THE BLOOD can be tested by methods which can only be performed in a laboratory.   The blood is never acid to litmus, but the degree of its alkalinity varies.   It is greater in men than in women and children ; it is diminished after violent exercise and the prolonged use of acids, and it is increased at the beginning of digestion, and after the prolonged use of alkalies.   In disease there is diminished alkalinity found with leukæmia, pernicious anæmia, anæmia, diabetes, cancer, great cachexia, poisoning with carbon monoxide and with acids, high fevers, and various toxic processes.   In chlorosis it is little if at all diminished.

The normal SPECIFIC GRAVITY of the blood is approximately 1055.   It may be estimated by mixing chloroform and benzol till the specific gravity of the mixture reaches 1055, and adding to this mixture a drop of blood from a pipette.   If the drop remains without rising to the surface or falling to the bottom the specific gravity of the blood is 1055.   If it sinks, continue to add chloroform drop by drop, shaking the mixture the while, until the drop becomes suspended.   If it floats, add benzol until the drop is suspended.   Then take the specific gravity of the mixture, and this is the same as that of the specimen of blood.

Significance of Altered Specific Gravity.—The amount of hæmoglobin can be estimated by finding the specific gravity of the blood, because it has been found that the specific gravity varies in proportion to the amount of hæmoglobin present, but this is of little practical value.   Sir Leonard Rogers examines the specific gravity of the blood in cases of cholera as a guide to the frequency and amount of injections of the hypertonic saline solutions which he has found so efficacious for that disease.   He employs a mixture of glycerine and water, which can be obtained in bottles, with full directions, from Messrs. Down Bros.   In the acute stage of cholera the specific gravity varies between 1060 and 1072 ; an injection is indicated when the reading is over 1065.

The FRAGILITY of red cells is tested by placing a drop of blood in salt solutions of strength varying by 0·04 per cent. and extending from 0·34 to 0·54 per cent.   When the red cells have settled a slight red tinge is seen above them in some tubes, whilst in others the red cells have completely hæmolysed.   Hæmolysis is said to start in the tube in which the first red tinge is seen and to be complete in that in which the red cells are all hæmolysed.   It is customary to test a normal person in a second set of tubes for comparison.   Normally, hæmolysis is absent in the 0·46 tube and complete in the 0·42 or 0·38 tube.   More accurate tests may be made by using more tubes with only 0·02 per cent. difference between each.   The fragility of the red cells is a diagnostic point in acholuric jaundice (§ 258).

SPECTROSCOPIC EXAMINATION of the blood.—The instrument chiefly used for clinical purposes is Browning's spectroscope.   It is used by holding up a glass containing a very dilute solution of blood, and looking through it at the light, or at a white cloud with a spectroscope placed between the blood solution and the eyes.   Hæmatoporphyrin (Fig. 114) has been found in the urine in sulphonal poisoning.   The discovery of methæmoglobin in the blood may be a means of warning the physician of the near onset of coma in diseases such as uræmia or diabetes.   Methæmoglobin is formed in nitro-benzol and potassium chlorate poisoning and other conditions (§ 30) ; carboxyhæmoglobin in coal gas poisoning ; sulphæmoglobin occurs rarely (§ 30).

The COAGULABILITY OF THE BLOOD is estimated by Sir A. E. Wright's coagulometer.[1]   It has a number of fine tubes, into which blood is drawn at definite intervals,

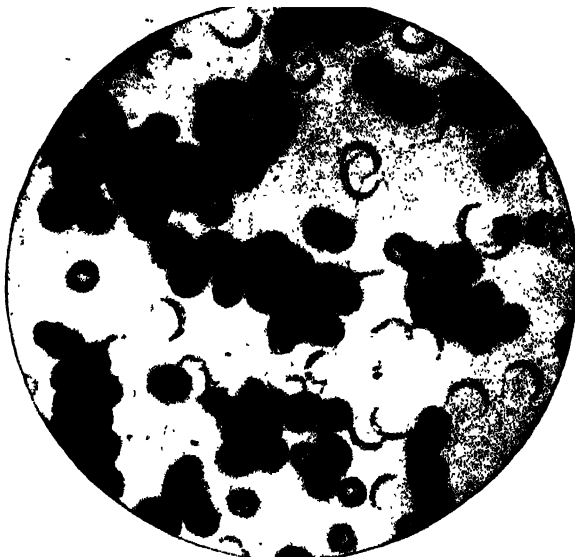[1] Hawksley supplies the instrument, with directions.

FIG. 113.—TRYPANOSOMA in blood of rat. Specimen prepared by Dr. Emilio Eccheverria; photomicrograph by Mr. Fred Clark.
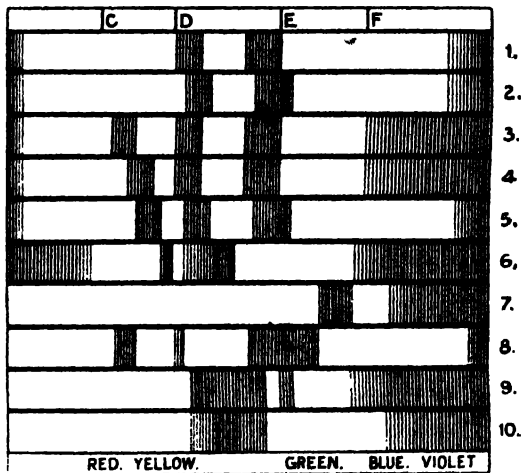

FIG. 114.—Drawn by Dr. Gordon Ward.

1. Oxyhæmoglobin—normal spectrum of fresh blood. 2. Spectrum of CO-hæmoglobin. Prepared by passing coal gas through normal blood. 3. Spectrum of methæmoglobin. Found in the blood of some cases of "Enterogenous cyanosis," in acute poisoning with various aniline derivatives and other drugs, and in the urine of Hæmoglobinuria. On the addition of Ammonium sulphide the spectrum changes to No. 10. 4. Spectrum of Sulph-hæmoglobin. Found in the blood in some cases of "Enterogenous cyanosis." It is not altered by the addition of small quantities of Ammonium sulphide. 5. The characteristic change produced in the spectrum of Sulph-hæmoglobin by passing through it coal gas. All the bands are shifted towards the blue end of the spectrum. 6. The spectrum of Hæmatoporphyrin in acid solution. Found in the urine in sulphonal poisoning, etc. 7. The spectrum of urobilin. This is a product of the destruction in the body of red blood corpuscles — i.e., of hæmolysis. It is found increased in the urine in many cases of Pernicious Anæmia, in Cholæmia, etc. 8. The spectrum of acid hæmatin. Produced by the decomposition of blood in acid solution. It is important to demonstrate this spectrum when it is desired to ascertain whether a given substance is blood. 9. The spectrum of Hæmochromogen or reduced hæmatin. Prepared by dissolving the suspected pigment in potassium hydrate and then adding Ammonium sulphide. The spectrum is easily demonstrated and very characteristic. 10. The spectrum of Hæmoglobin, sometimes called "reduced Hæmoglobin." The colouring matter of blood deprived of Oxygen: it exists in venous blood. Nos. 8 and 9 are important in medico-legal tests. •

and at varying times the operator blows down the tubes. When the blood cannot be blown out it has coagulated. The coagulation time is thus readily calculated. Coagulation depends upon the presence of lime-salts and fibrinogen, which are contained in the blood plasma, and of nucleo-proteid, which is contained in the substance of the leucocytes and blood platelets. The nucleo-proteid is liberated when the leucocytes are disintegrated, and this may occur when infective toxins circulate in the blood, when the blood-vessels are diseased, and in cachectic states, and thus thrombosis may in some cases be explained. Coagulation within the body is also hastened by the addition of certain salts, such as calcium chloride. Coagulation within the body is diminished in certain infective diseases, and in urticaria, prurigo, and allied erythematous conditions, and the administration of calcium chloride in 20-grain doses, as the author has shown,[1] is of great use in alleviating prurigo and urticarial conditions.

The FREEZING-POINT OF THE BLOOD is normally 0·56° C. It is lowered to 0·60° C. in cases of renal disease, in which an excess of urea and salts is present, and reaches 0·8° C. in cases of impending uræmia.

PART C. DISEASES WHICH GIVE RISE TO GENERAL DEBILITY, WITH OR WITHOUT ANÆMIA AND EMACIATION : THEIR DIAGNOSIS, PROGNOSIS, AND TREATMENT.

§ 431. Routine Procedure and Classification.—Here, as elsewhere, we have three points to investigate :

First, the LEADING, and perhaps the only SYMPTOM complained of by the patient will be debility, or pallor of the skin, or loss of flesh.

Secondly, the HISTORY OF THE ILLNESS, its date, and mode of onset and mode of evolution. Often these data are vague, but special inquiries should be directed to the condition of the digestion in times past, and any other points relating to nutrition.

Thirdly, the PHYSICAL EXAMINATION of the patient, commencing with that physiological system to which the results of our previous inquiries have directed attention, and then going through all the systems seriatim. An examination of the blood should be made in all anæmic or doubtful cases—viz., cell-counts, hæmoglobin, and films.

Classification.- If anæmia is suspected or PALLOR OF THE SKIN is the leading symptom, turn first to Group I., below.

If LOSS OF FLESH is most prominent, turn to Group II., p. 631.

If GENERAL DEBILITY (without obvious pallor or loss of flesh) is most prominent, turn to Group III., p. 637.

GROUP I. ANÆMIC DISORDERS

Anæmia is an alteration in the composition of the blood, the leading character of which is a deficiency in the hæmoglobin. The disorders giving rise to **pallor of the skin** are :

[1] **The *Lancet*, August 1, 1896.**

COMMONER.

I. Chlorosis (primary anæmia), § 432.
II. Pernicious (Addisonian) anæmia (§ 433).

Constitutional Conditions.

III. Constitutional syphilis (§ 434).
IV. Lead poisoning (§ 435).•
V. Tuberculosis.
VI. Carcinoma.

Visceral Disorders.

VII. Gastro-intestinal conditions.
VIII. Chronic aortic disease.
IX. Chronic renal disease.
X. Chronic hepatic disease.

Recognised by History.

XI. Hæmorrhage, hyperlactation, and other causes of long-continued drain.
XII. Post-febrile anæmia.
XIII. Chronic suppuration, septic processes, and lardaceous disease.

RARER (*in this country*).

With Signs in Spleen and Lymphatic Glands.

I. Leukæmia (§ 437).
II. Hodgkin's disease (§ 438).
III. Splenic anæmia (§ 439).
IV. Acholuric Jaundice (§ 258).

With Signs referable to Skin.

V. Scurvy (§ 440).
VI. Hæmophilia (§ 441).
VII. Addison's disease and other maladies mentioned in Groups II. and III.

With History of Residence Abroad.

VIII. Malaria and other tropical and parasitic diseases.

Anæmias of Infancy.

I. Primary anæmias.
II. Secondary anæmias.
III. Congenital anæmia.
IV. Infantile scurvy.
V. Anæmia splenica infantum.

The pallor may present to the experienced observer a difference in kind and degree in the several affections. Thus, the greenish-yellow colour of chlorosis, the lemon-yellow of pernicious anæmia, the earthy tint of carcinoma, the sallowness of aortic disease and interstitial nephritis, the pasty white of parenchymatous nephritis, and the transparent waxy look of lardaceous disease are very suggestive to the careful observer. The microscopic examination of the blood also reveals differences which are mentioned below. The age of the patient will often give us a valuable clue.

The blood shows marked **diminution of hæmoglobin** *and other changes, and there is* NO DISCOVERABLE PRIMARY ORGANIC LESION. The disease is probably CHLOROSIS or PERNICIOUS ANÆMIA ; the age and sex of the patient and the colour-index of the blood being very different in the two diseases.

§ 432. I. **Chlorosis** (" green sickness," " poverty of blood ") is a chronic malady in which the main feature is a diminution of the colouring matter of the blood, unaccompanied by any gross lesion, not preceded by any primary or causal disease, and occurring mostly in young women. This disease is now much less frequently encountered than formerly.

The chief *Symptoms* are (1) failing strength of vague onset and considerable duration, accompanied by pallor of the surface, but unaccompanied by wasting. There is, indeed, often an excess of adipose tissue. The pallor is marked in the lips, gums, and conjunctivæ (as may be observed by pulling down the lower lid), and the sclerotics have a bluish colour.

The skin sometimes presents a greenish hue, hence the name chlorosis (χλωρός, green). These patients generally present themselves on account of either cardiac or digestive symptoms, headache, or amenorrhœa. (2) Cardio-vascular symptoms, such as dyspnœa on slight exertion, palpitation, and a tendency to syncope. In a fair proportion of the cases enlargement of the area of cardiac dulness can be made out. Hæmic murmurs are heard, especially over the pulmonary area. In marked cases the " bruit de diable " is to be heard—a continuous hum heard when the stethoscope is gently placed over the jugular vein in the neck. Œdema of the ankles at night is common, and venous thrombosis may be met with.

Hæmic or Anæmic Murmurs may be either systolic or double in rhythm, never diastolic alone or presystolic. They are usually soft and blowing, but may be extremely loud and rasping ; loudest in the pulmonary area, but they may be heard all over the precordium, very rarely in the axilla ; often louder when the patient is lying down or has rested, and apt to vary from day to day.

(3) Disturbances of digestion are generally present, such as deficient or capricious appetite, and discomfort or even vomiting after food ; and there may be atonic dyspepsia, gastric atony, and gastroptosis. Intractable constipation often precedes and accompanies the affection. (4) Symptoms referable to the nervous system, such as headache, neuralgia, tinnitus, vertigo, defective attention, nervousness, irritability or depression of spirits, spots before the eyes. Vasomotor signs are frequent ; a tendency to " dead fingers," morbid flushing and variable temper. The appetite may have strange cravings, as for vinegar and slate pencils. (5) Amenorrhœa is usual, dysmenorrhœa not infrequent ; menorrhagia rarely accompanies chlorosis. The thyroid may be enlarged, and other symptoms of hyperthyroidism are not uncommon. (6) Some authorities say that chlorosis may be attended by elevation of temperature from time to time, but adequate proof in the post-mortem room should be forthcoming that such cases have not been due to some undiscovered syphilitic, tuberculous, or septic lesion (*vide* p. 64, footnote). In ordinary cases the temperature is subnormal. (7) *Blood changes.*—There is often but little diminution in the number of red corpuscles, though a great diminution in the hæmoglobin which they contain. The blood appears pale, and the total hæmoglobin is sometimes as low as 30 per cent. of the normal. In severe cases, however, there is also a considerable diminution (down to 2,000,000) in the number of red corpuscles. Poikilocytosis, polychromatophilia and nucleated red cells are uncommon except in severe cases (see § 428). There is an excess of the watery constituent of the blood or " hydræmic plethora."

The *Diagnosis* is not, as a rule, difficult, by reason of the age and sex of the patient. The pallor of the skin, though it may have a yellowish-green tint, is usually quite different from that of jaundice or the cachexia of malignant disease. It has at times to be differentiated from that due to any of the conditions mentioned in the succeeding sections below,

The chief danger is lest an early stage of chronic tuberculosis should be overlooked ; and to avoid this a thorough examination of all the organs should be made, the principal physical signs in chlorosis being the murmurs and the pallor. The pallor of renal disease is an ivory white, and is attended by albuminuria, tube-casts, and in some forms by generalised dropsy. Chlorotic girls may have œdema of the ankles, but there is no albumen in the urine. The diagnosis from organic heart disease is given under Cardiac Murmurs (§ 37).

Prognosis.—The disease is rarely fatal, but it is extremely liable to relapse, and the symptoms are sometimes so grave as to necessitate strict confinement to bed. It is essentially a chronic condition, and its course always extends over many months, unless checked by treatment. Recurrences are common. Gastric ulcer may follow. Thrombosis of the femoral vein may occur, and, occasionally, of the longitudinal sinus. Optic neuritis may occur with recovery, but ocular paralysis and proptosis, due to intracranial thrombosis (Hawthorne), are usually fatal signs. " Spurious hæmoptysis "—*i.e.*, hæmoptysis without any pulmonary mischief—probably coming from the mouth or throat, occurs occasionally.

Etiology.—Apart from relapses, the disease is practically confined to young women between the ages of fifteen and twenty-five, *i.e.*, it occurs during the establishment of the changes in the organism which are initiated at puberty. Frequently there is a history that the mother or sisters have been similarly affected, and patients are often members of comparatively large families. It is a curious and unexplained circumstance that more blondes than brunettes are affected. The disease affects both rich and poor. The familial incidence, the onset at puberty and the richness of the plasma, all support the suggestion that it is due to some disturbance of the balance between the internal secretions.

The *Treatment* of chlorosis consists, first, in rest in bed ; secondly, in the administration of iron ; and thirdly, in attention to hygienic measures and the treatment of symptoms. As in all anæmias, the tonic effect of sunlight and fresh air cannot be insisted upon too much. Complete rest is advisable for three weeks at the beginning of the treatment ; strict confinement to bed shortens the course of the disease. It is well to remember that at least three months are required to effect a cure, and the patient should be warned of the danger of relapse. Iron should be administered in sufficient quantities and in gradually increasing doses (*e.g.*, one Blaud's pill, F. 86, thrice daily for the first week, two for the second, three for the third, and so on for five weeks, then decreasing the dose). Blaud's pills must be freshly made, or they may pass unaltered through the bowel. Liq. ferri perchlor. (m. 5 to 10 (0·3–0·6)) is a very valuable remedy, if the stomach will tolerate it. Constipation must be specially guarded against. Iron may be combined with magnesium sulphate. Ferri sulphas (alone or with aloes) gr. 1 (0·06) thrice daily for the first week, increased to gr. 2 (0·13) for the second and gr. 3 (0·2) for the third, continuing at gr. 9 (0·6) per diem for three months, will seldom fail to effect a cure. If

nervous symptoms be present, syrupus ferri phosphatis is useful; while ammonio-citrate or peptonate of iron or reduced iron are excellent, especially for patients with delicate digestion. When menorrhagia is present, treatment directed to this condition should be adopted (§ 348). If the case resists iron and arsenic in one or another form, the diagnosis should be revised, and tubercle or one of the other conditions mentioned below considered. Other remedies include arsenic, Levico water (which contains iron and arsenic), cod-liver oil, lacto-phosphate, malt extract, manganese preparations, dilute acids, and the administration of oxygen. Intramuscular injection of iron is much in vogue on the Continent, and good results are reported therefrom. "Late hours, tight lacing, overstudy, worry, and idleness are to be forbidden. To enlarge the capacity of the chest prescribe respiratory exercises. The diet must be liberal, especially in regard to nitrogenous food in the form of butcher's meat (which should be taken at least once daily), and milk. Green vegetables and fruit are useful. The patient should avoid drinking much at meal times; half a tumbler of fluid is sufficient. Two to three hours after food a tumbler of hot water may be taken. The food should be carefully masticated, and the teeth attended to if necessary.

§ **433.** II. **Pernicious Anæmia** (Synonyms : Idiopathic Anæmia of Addison, Addisonian Anæmia) is a relatively rare variety of primary anæmia first described by Addison (who called it idiopathic anæmia), and now known to be associated with certain other signs of toxæmia and of hæmolysis, chiefly affecting men in the second half of life, and running a chronic and generally fatal course. The principal changes constantly present after death are : The heart exhibits fatty degeneration ; the spleen is engorged ; the bone-marrow is unduly red, and contains a great number of nucleated red cells, especially gigantoblasts ; and the liver is fatty, sometimes enlarged, and contains an excess of iron deposited within it, as shown by the ferrocyanide reaction. Extensive atrophy of the gastric mucosa and achlorhydria may also be found. Changes have been discovered in the sympathetic ganglia, and in the spinal cord.

The *Symptoms* may be divided into two groups : (1) Those due to anæmia *per se*, and (2) those peculiar to " pernicious " anæmia. Among the first groups are : General weakness and anæmia of insidious onset, with their usual effects—palpitation, dyspnœa, a tendency to syncope, hæmic murmurs, and other symptoms as in chlorosis (*q.v.*). There is little if any wasting ; there may, indeed, be much subcutaneous fat. Among the second group are : (1) General toxic symptoms, such as lassitude, irregular pyrexia from time to time (though the temperature at other times may be normal or subnormal). (2) Gastro-intestinal attacks with abdominal pain, comparable in severity to the crises of tabes. Diarrhœa may resist treatment, then suddenly cease with improvement of other symptoms. The tongue is sore, due to an atrophic gastritis ; this may be the first symptom of the disease, and the patient may be able from its presence to foretell relapses. (3) Nervous symptoms, varying from slight ataxy to paralysis of all the limbs, due to sclerosis of the spinal cord. Sometimes these precede the anæmia and occasion difficulty in diagnosis. (4) Symptoms due to hæmolysis—*e.g.*, anæmia, with urobilinuria, excess of uric acid in the urine, and a lemon tint of the skin. (5) There is a marked tendency to hæmorrhage into the retina, sometimes into the skin, and from the mucous membranes into the internal organs. (6) *Blood changes* (§ 428, Fig. 110). (*a*) The red corpuscles are much reduced in number, often falling to less than one-fifth of the normal. (*b*) The colour-index is high—*i.e.*, the percentage of hæmoglobin in each corpuscle is increased, but the total hæmoglobin in the blood is diminished—*e.g.*, it may be as low as 8 to 15 per cent. of the normal. (*c*) The

most frequent alteration in the blood consists of irregularity in size (megalocytes and microcytes) and shape of the red corpuscles (poikilocytosis) ; nucleated red corpuscles (megaloblasts and normoblasts) are present. (*d*) Leucocytosis is not present ; leucopenia may be extreme.

The *Diagnosis* is based on a consideration of the symptoms, the age and sex of the patient, and the blood changes—viz., a marked alteration and diminution in the red corpuscles without diminution in the hæmoglobin value of each cell—the converse of chlorosis. Although this condition of the blood is most often met with in association with pernicious anæmia, it must be remembered that it may also occur in other hæmolytic anæmias (*e.g.*, cholæmia). The diagnosis from severe secondary anæmias should not be difficult when the symptoms above described as peculiar to pernicious anæmia are present. Difficulty arises when gastro-intestinal and nervous symptoms are absent. In such cases a definite diagnosis may be impossible. Although Addisonian anæmia may be closely simulated by severe septic anæmias, the latter usually have a low colour-index and high rather than low leucocyte count.

Prognosis.—The disease is slow but progressive, and almost invariably fatal. Progress may be estimated by examining the blood from time to time. The main complications are cardiac weakness, vomiting and diarrhœa, visceral hæmorrhages, cerebral or spinal hæmorrhage, and degeneration in the spinal cord, chiefly affecting the posterior columns. Headache, nervousness, and prostration are fairly constant, but the intellect is usually clear to the end ; sometimes convulsions and coma occur. As recovery from one attack is almost always followed by a relapse it is important to take care that in the interval between the attacks treatment puts the patient in good condition. Then the relapses may become less severe and finally cease. Each attack may deprive the patient of half to three-quarters of the blood in his body ; this is not fatal if at the beginning of each relapse the patient has a normal amount of blood.

Etiology.—Pernicious anæmia chiefly attacks males from twenty-five to forty-five, occasionally women of the same age ; it is very rare in the young. It comes on insidiously, without apparent cause. Hunter believes the disease is due to a gastro-intestinal infection, with absorption of toxins, which destroy the red corpuscles (hæmolysis), and thus give rise to deposition of iron in the liver, urobilinuria, anæmia, and other symptoms. It is necessary to say something about the connection of oral sepsis with this disease, as this is very generally misunderstood. The disease is probably due to a specific micro-organism and the frequent association of a sore tongue (not of oral sepsis) with its onset, and the almost invariable presence of atrophy of the alimentary mucous membranes suggest that the site of the invasion is in these positions. It is not disputed that great destruction of blood follows the onset of the disease ; to this the blood-forming organs respond by great increase of activity. Now the presence of any septic focus from which the products of the common pyogenic organisms can be absorbed is a serious hindrance to this activity, because these products exercise a depressing influence on the blood-forming organs, leading to varying degrees of *atrophic* changes. Such a focus associated only too often with this as with many other diseases, is to be found in the mouth, either in the form of dental caries, apical abscesses or pyorrhœa alveolaris.

Treatment.—Complete rest in bed is necessary. In order to give the blood-forming organs every assistance in their endeavour to make up for the blood destruction it is of the first importance to remove all septic foci. For this reason also it is important to remove not only oral sepsis but sepsis wherever found. We have no known method of reaching or destroying the specific organism which is conjectured to cause the disease, but by improving the power of resistance of the body, especially between the attacks, much may be done (*cf.* Prognosis). The diet should be nutritious and rendered digestible. The stomach must not be overloaded. Arsenic has a greater control over the disease than any other drug, but it has often been noted to be of more use in the primary attack than during a relapse ; the dose should be gradually increased until a drachm of Fowler's solution is being given daily. It may be administered

hypodermically as the cacodylate of soda gr. ⅓ to 1 (0·03–0·06), or in the form of atoxyl gr. ⅓ (0·05). Naphthol, salol, and other intestinal antiseptics have been used with advantage. Red marrow, transfusion of blood, oxygen inhalations, and subcutaneous injections of antistreptococcic serum have also been tried, and have in some cases coincided with marked improvement. Splenectomy is only of temporary benefit, and should not be performed. For the acute attack purgatives are of more importance than any other medicament. If the tongue is very sore 5 per cent. cocaine in glycerine may be prescribed and applied with the finger.

Aplastic Anæmia is a disease in which the bone-marrow loses its power of forming blood corpuscles. This is apparently due to the action of bacterial toxins, but certain cases are idiopathic in the sense that no causative bacterial infection is discoverable. It has followed exposure to the more penetrating rays of radium and X-rays. The *Symptoms* are those of a profound toxæmia. There is an especial liability to hæmorrhages and a marked stomatitis. • The blood shows an advanced degree of anæmia which differs from that seen in any other condition by the absence of regenerative forms, including megalocytes, polychromatophilic and granular red cells. The decrease in leucocytes leads to a relative lymphocytosis ; the lymphocytes may reach as many as 95 per cent. of the total white blood corpuscles. The *Diagnosis* can only be made by a careful examination of the blood. The *Prognosis* is uniformly bad, and the disease is usually rapidly fatal, but recovery has been known.

The patient is PALE, *but the anæmia does not quite conform to the preceding types, and has* **not been readily amenable to treatment by iron.** The disease is probably some LATENT CONSTITUTIONAL CONDITION (syphilis, plumbism, tubercle, or carcinoma), or some LATENT VISCERAL DISEASE.

§ **434.** III. **Syphilis** (Synonym : Hunterian or Constitutional Syphilis) is a constitutional malady, due to a Spirochæte infection, which starts as a superficial ulcer (chancre) at the seat of inoculation, runs a prolonged and indefinite course, is liable to break out anew during the whole lifetime of the patient without fresh infection, even after many years of quiescence, and in its later stages produces serious cardio-vascular degeneration and granulomatous deposits throughout the body, particularly in the skin and nervous system.

Symptoms. -- Syphilis leads to a degree of pallor which may simulate chlorosis or other forms of anæmia very closely. When no history of primary syphilis is obtainable, and no physical signs can be discovered, the diagnosis from other forms of anæmia may be difficult. For the sake of convenience, the symptoms of syphilis are divided into three stages, but it must not be forgotten that their mode of appearance is extremely variable, and that the three stages may even appear simultaneously in certain patients. *Primary Stage.*—The period of incubation generally lasts about *three weeks*, but it may vary from ten to forty-six days. The initial manifestation appears as a superficial ulcer (the hard or Hunterian chancre) at the site of inoculation. It is usually single, and occurs most commonly on the prepuce or glans penis in the male and the labiæ and nymphæ in the female. It originates as a flat, elevated, painless papule, which slowly enlarges, and may desquamate without breaking down, or superficial erosion or ulceration takes place with a slight serous discharge, thus differing from the deep excavated ulcer of the " soft sore " or non-

Hunterian chancre.   The underlying induration of the tissues is always
a marked feature, hence its name " hard sore."   The lesion after a time
cicatrises, and usually leaves behind it some slight discoloration or indura-
tion, or both, which mark the site.   Sometimes the primary sore is so
slight as to be overlooked, and appears to be wanting, especially in the
female.   About the same time, or within one or two weeks, the associated
lymphatic glands, usually in the groin, become enlarged and hardened.
Even thus early in the disease, the red blood discs may be diminished to
3,000,000 per cubic millimetre or even less, and there is pallor and weak-
ness, conditions which increase if the disease is untreated.   This glandular
enlargement may become generalised, and may persist for months or
years, and hardness of the lymphatic glands may thus serve as an aid
to diagnosis at any time.

The *Secondary* symptoms make their appearance about three weeks
after the first appearance of the chancre (four to twelve weeks after inocu-
lation).   In typical cases a faint generalised dusky macular rash (which
may be brought out more distinctly by a warm bath) appears chiefly on
the chest and abdomen, nearly always attended by sore throat, and often
attended by malaise, pains in the limbs, anæmia, and slight pyrexia.   The
rash takes about three weeks to mature and three weeks to decline.   The
pyrexia, which is generally overlooked, has already been described (§ 412).
The sore throat is usually of an indolent, ill-marked kind, with whitish
secretion resembling snail tracks (§ 132).   The eruptions which may appear
now and hereafter are of many different kinds--macular, papular, scaly,
pustular, tubercular, practically never eczematous or vesicular.   The
characteristics of these (see also § 520) are their reddish-brown colour,
generalised or symmetrical distribution, grouping in segments of circles,
and their preference for the forehead and flexor surfaces, their poly-
morphism and absence of itching.   The hair may fall out, and the nail-beds
be affected with an indolent inflammation.   Moist " mucous patches,"
with a highly contagious secretion, are apt to appear at the corners of the
mouth and other mucous orifices.   The diagnosis of the skin symptoms
(§ 520) and the lesions of the mucous membranes (§ 172) are dealt with
elsewhere.   The eyes may become affected by iritis, choroido-retinitis,
and the bones with periostitis in which the pain is worse at night, and
the joints with synovitis.   Any of these symptoms may crop up again
and again during the ensuing months or years.

Later Stages (so-called tertiary symptoms) and *Varieties* of syphilis.—
In practice it is convenient to recognise two broad varieties of syphilis.
In most cases of a BENIGN type, *adequately treated*, there is no recurrence
of symptoms after the second stage above described ; in short, there is
no tertiary stage.   But in other cases the disease assumes a MALIGNANT
type either by reason of the intensity of the virus or the predisposition or
debilitated state of the individual, combined perhaps with inadequate
treatment in the earlier stages, and such cases are characterised by severity
of the initial symptoms and a tendency to recurrence at intervals through-

out life. It is in such cases more especially that one meets with what are known as "tertiary" symptoms. Malignant or tertiary lesions, as exemplified, for instance, in the skin, are characterised by having a greater and deeper infiltration, a greater proneness to suppuration, ulceration, and scarring, and by being followed by more loss of tissue than the benign lesions. All the same skin symptoms noted in the secondary stage may recur, but they are more apt to be localised and asymmetrical in distribution, serpiginous in outline, lenticular or nodular in shape, and pustular or ulcerating in character than the corresponding secondary symptoms. Nodular or infiltrating gummatous deposits followed by scarring, and perhaps by ulceration, may affect the mucous membranes, particularly in the oral cavity and its diverticula, the liver and other abdominal organs, and the cephalic and the genito-urinary organs, and lead to fibroid degeneration, stricture, or destruction of the proper tissues and functions of the parts. The bones are often attacked by gummatous periosteal deposits, leading in the case of the hard palate to perforation, and in the other flat and the long bones to the formation of "nodes." An intermitting pyrexia may accompany the formation of gummata. The arteries may become thickened and arterioles blocked; partly for this reason, partly by the proneness of the virus for the nerve-tissues and meninges, the nervous system is specially apt to be involved. But even this list does not complete the account of this insidious, prolonged, and terribly far-reaching disease, for it is, as mentioned in previous chapters, one of the two causes of lardaceous disease of the liver, spleen, kidneys, and intestines. More or less anæmia is a symptom throughout the disease, and in untreated or malignant cases of syphilis the cachexia may sometimes be fatal, as in patients referred to on p. 609 and elsewhere.

PARASYPHILIS ($\pi\alpha\rho\alpha$ = derived from) was a term formerly applied to certain diseases which were considered to be due not to definite syphilitic deposits, but to the indirect effects of the syphilitic poison, or to its after-effects. Thus Locomotor Ataxy and General Paralysis of the Insane were called parasyphilitic diseases; but recent work has revealed the presence of the spirochæte in the nervous system.

HEREDITARY or CONGENITAL SYPHILIS.—We have seen how ubiquitous the consequences of syphilis may be, but it is upon the children of such parents that the heaviest nemesis falls. Happily, the mother very often aborts, syphilis being one of the commonest causes of abortion, or the child dies and decomposes within the uterus, or, being born alive, it dies in the first twelve or eighteen months of life of marasmus or its complications. Thus a series of miscarriages or stillbirths, or a heavy mortality among children in the early months of life, imply a strong probability of syphilis in the parent. If the child be born alive the primary chancre is of course wanting, but the symptoms confirm more or less to the secondary symptoms above described. The infant is sometimes healthy at birth, but in a few weeks it develops "snuffles," or a ham-coloured eruption on the buttocks, flexures, palms, or soles, and marasmus sets in and

is followed (if active treatment is not adopted) by any of the other secondary symptoms above mentioned. The child is fretful, the cry is hoarse, and the bones are tender, and gastro-enteritis, bronchitis, or pneumonia may complicate matters. If the child survives the first twelve or eighteen months of life, a long period without fresh syphilitic manifestations ensues, excepting perhaps in the rapid decay of the temporary teeth and stunted growth of body. About the seventh year, however, the permanent teeth appear, and usually present the pegged shape and notched border described by Sir Jonathan Hutchinson. Again, there may be an interval of quiescence, but about the fourteenth year of puberty, interstitial keratitis, deafness, periostitis, or synovitis may appear, the skin, viscera, and nervous system only rarely being affected. From this time onwards the evidences of hereditary syphilis consist of the consequences of the previous lesions on the general development, the skin, the mucous orifices, the malformation of the bones, the eyes, ears, and teeth, which are summarised in Table XXIV., p. 610.

The *Diagnosis* of the Hunterian chancre will be found in surgical works. The diagnosis of syphilitic symptoms and lesions in the skin, nervous system, liver, and other parts, will be found in the appropriate chapters of this work. The existence of hard shotty glands as an aid to diagnosis has already been referred to. In regard to the diagnosis of syphilitic from other forms of anæmia undoubtedly mistakes may easily be made. I remember the case of a very anæmic lad of fifteen in whom the only other symptom besides those of anæmia was a slight rise of temperature in the evening; after death, however, gummatous deposits were found in the meninges in the frontal region and elsewhere. A somewhat similar case of gumma of the liver was reported by the late Dr. J. S. Bristowe. Syphilis should always be suspected in obscure cases of anæmia, and very often the amenability to treatment by iodide and mercury will be a revelation. The presence or history of an eruption should be noted, and the viscera, the bones, and the eyes very carefully examined. The Wassermann test for syphilis (§ 667), is quite reliable, if correctly performed. The practitioner should send a sample of the blood to a well-equipped laboratory, as the diagnosis of syphilis is of the highest importance to the individual and to the community.

Prognosis.—Syphilis is never fatal (except, perhaps, in the case of the fœtus) by the intensity of its toxæmia, like small-pox or scarlatina; but in infancy it may cause a fatal marasmus. In adults it only kills, usually after a life of invalidism, by its complications or by involving some vital part. Benign cases of the disease adequately treated, if the patient lives a temperate, hygienic life, may give no after trouble, and many such persons live to old age and have perfectly healthy children. Nevertheless, it behoves even these patients to be constantly on their guard, for once syphilitic means that they are always liable to the possibility of recurrence even to the end of their days. Malignant types of the disease are sure to recur, and to require active treatment on and off throughout life. The

C.M.   •   R R

severity and duration of an attack of syphilis are influenced by a number of circumstances, some of which are hard to gauge. The habits and mode of life (especially as regards intemperance), age, occupation, exposure, privation, pre-existing disease (especially tuberculosis and renal disease), all doubtless influence the course of the malady. The disease is often said to prevail in a particularly virulent form in some naval and military stations. But of all factors, the one which influences the prognosis of syphilis more than anything else is adequate and continuous treatment during the earlier phases of the malady.

TABLE XXIV.—HEREDITARY SYPHILIS.[1]

A. INFANTILE MANIFESTATIONS (three weeks to three months).

I. May be born quite healthy. Then symptoms resembling acquired secondary syphilis appear—symmetrical, transitory, etc.

II. Mucous Membranes { Snuffles.
{ Condylomata around anus or mouth.

III. Marasmus, leading to " senile aspect " ; very marked wasting, often fatal.

IV. Skin { Papular
{ Scaly
{ Pustular
{ Bullous
{ Polymorphic } Always symmetrical, transitory, ham-coloured; on buttocks because of urine and fæces ; in flexures because of perspiration. Patches of peeling erythema about face, nates, neck, etc.

V. Iritis.

VI. Definite Periostitis—Tenderness of bones and " rheumatic " pains, epiphyseal abscesses, or caries of long bones. Skull—thinning in one place, thickening in another. Skeletal deformities and nodes.

B. ADOLESCENT MANIFESTATIONS (commencing about puberty).

Which come on after an interval of quiescence of some years, if the child survive the first years of life.

I. Nebular Keratitis—first one cornea, then the other appears like ground-glass—between tenth and twentieth year. Ultimately quite clears up under treatment.

II. Deafness—between puberty and twenty-third year—comes on with noises in ears—but without pain or otorrhœa—terminates in recovery or complete incurable deafness.

III. Periostitis of long bones (rarely skull)—generally causes overgrowth, sometimes bending, or nodes, occasionally suppuration.

IV. Synovitis (painless)—knees or other large joints.

V. Skin, viscera, and nervous system rarely affected at this stage.

C. LATE MANIFESTATIONS (from fifteen years upwards).

NOTE.—All of these, being the result of infantile syphilitic inflammations, are absent if syphilitic manifestations have been previously wanting.

I. *Constitutional Effects.* { *Infantile build.*
{ *Retardation* of development, of growth, of dentition, of the catamenia.

[1] This table is after Fournier, modified.

Skin—Peribuccal cicatrices radiating from the mouth; Parrot's cicatrices.

II. *Tegumentary System.*
    Eruptions (very rare)—Lupoid ulceration, gradually spreading, may appear.
    Mucous membranes—Cicatrices, of the throat, palate, and round the mouth. Hole in palate, etc.

III. *Osseous System.*
    Cranial malformations—prominent frontal eminences, natiform cranium, asymmetry, hydrocephalus.
    Nasal malformations—" Duck-nose," depressed septum, " opera-glass nose."
    Tibial deformities—" Sword-blade " tibia ; or curving with shortening ; or increased length.
    Joint lesions—Chronic painless effusions, and distorting arthropathies.

IV. *Hutchinson's Triad.*
    1. Eye
        Ocular malformations.
        The remnants of interstitial keratitis (striæ in cornea), iritis, or choroidal atrophy.
    2. Ear . Cicatrices of the tympanum, deafness.
    3. Teeth
        Underhung or displaced jaws, irregularities or absence of teeth.
        Dental dystrophies—microdontism, amorphism, " pegged teeth " of Hutchinson (Fig. 3).

V. *Family History.*
    Miscarriages and stillbirths in series.
    Heavy mortality among children in first three months of life.

Etiology.—The specific organism is a feebly staining spirochæte, to which the name *Spirochæta pallida* - Schaudinn (*Treponema Pallidum*) has been given. It can be obtained not only from the primary sore, but in abundance from condylomata, and also from the viscera in secondary, tertiary, and congenital syphilis. The organism has a corkscrew shape with from eight to twelve curves ; one end is filamentous, the other thicker and more deeply staining. It is differentiated from a commonly occurring spirillum, the *Spirillum refringens,* in that the latter has fewer and less delicate curves, stains equally deeply at both ends, and has no thickening at one end.
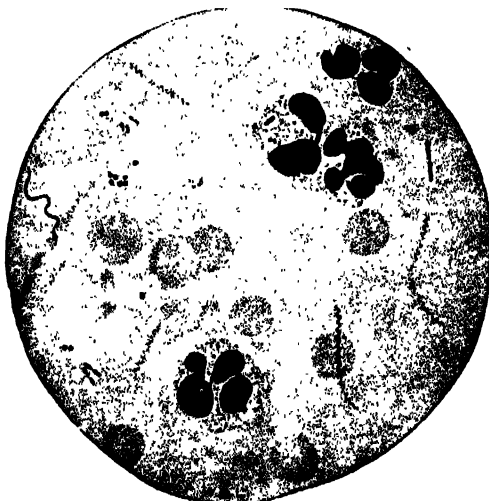

FIG. 115.—SPIROCHÆTA PALLIDA (TREPONEMA PALLIDUM) of SYPHILIS, magnified about 500 diameters. Illustration lent by the courtesy of Colonel Sir W. B. Leishman, R.A.M.C. The organism is of a spiral form like a long corkscrew. The wavy organism on the left is the *Spirillum refringens.*

Syphilis resembles the specific fevers in having a period of incubation

followed by a characteristic eruption. One attack renders a person immune to a second attack, except in cases where the first was cured by salvarsan. It differs from other specific fevers in the extreme length of its course, which may last many years, in the long intervals which may separate its various manifestations, and above all in its liability to recur without fresh infection. Inoculation can only take place through an abrasion of the skin or mucous membrane, and may occur in three ways : (a) Usually it is by direct contact with an infected person, generally during sexual intercourse, but in some cases (e.g., in doctors and midwives) as the result of examining diseased persons, by suckling (as in wet nurses), kissing, etc. ; (b) occasionally by the use of contaminated articles—e.g., spoons, cups, pipes, towels, surgical instruments, or (?) the seat of a w.c. In the first two stages the blood and the *moist exudations of all the lesions are certainly contagious.* In the later stages some difference of opinion exists as to the contagiousness of the blood and secretions. My own experience is that all moist lesions in all stages (and therefore the blood also) are contagious, and I well remember a patient of mine who had contracted the disease thirty-three years before, and who conveyed the disease to his sister by kissing, he having at the time only a small fissure at the angle of his mouth. In regard to marriage, I see no reason to modify the rule about to be stated, however old-standing the disease may be. The use of human vaccine lymph, even when free from blood, for vaccination purposes from arm to arm, was undoubtedly the occasional means of propagating syphilis, but the frequency of the occurrence was certainly exaggerated. (c) To the offspring syphilis may be conveyed by hereditary transmission of the virus from either parent. If, as frequently happens, the mother becomes infected shortly before she becomes pregnant, or during the early months of pregnancy, the child seldom escapes the disease ; if after the seventh month, however, the child may be healthy. It is said that the child may be infected from the father, and that the mother may nevertheless escape ; but this is extremely doubtful, for under these circumstances the mother never contracts the disease from her child after its birth (Colles' law). The date when marriage is permissible is a most important one. It should under no circumstances be sanctioned within two full years after infection, even in the mildest case. If the patient has been free from any syphilitic symptoms and the Wassermann test remain negative on three successive occasions and also after a provocative injection of salvarsan, there is every prospect that the offspring will be healthy.

Syphilis has, there is little doubt, prevailed from very ancient times, and has occasionally occurred in the form of widespread and severe epidemics, particularly when introduced into previously healthy communities. Individuals of all races and ages are subject to it. Metchnikoff and Roux and Neisser succeeded in inoculating apes with syphilitic virus and reproducing the disease in them ; the anthropoids are more readily infected, and show more characteristic lesions than the lower apes.

Extremes of temperature seem to intensify its virulence. Tuberculous and otherwise debilitated subjects usually incur syphilis in a severe and malignant form.

Treatment should be begun the moment the diagnosis is made. The most successful results are obtainable when treatment is commenced before the secondary symptoms appear. In mercury, iodide of potassium and salvarsan we have powerful remedies for controlling this serious and far-reaching disease. When Ehrlich introduced dioxy-diamido-arseno-benzol (salvarsan or " 606 ") in 1910, it was believed that one injection would cure syphilis. Present evidence tends to show that if administered very early the secondary stage may be aborted. Relapses certainly occur when the drug or its substitutes are given in the late secondary stage. The findings of the Royal Commission on Venereal Disease awakened the public to the gravity of the ravages wrought by syphilis when imperfectly treated, and legislation has been enacted to bring free and efficient treatment within the reach of all sufferers. Kharsivan, arsenobillon, novarsenobillon, diarsenol and galyl are recommended arsenic compounds. They are best administered intravenously— a method to be attempted only by those who have studied its technique. The patient should have no food for a few hours before, and should go to bed after the injection. Six or eight weekly doses may be required, and it is advisable to take mercury during this and any subsequent course.

These drugs should never be given when arterial, nerve or kidney disease is present, lest blindness, deafness, or some fatal accident ensue. The fever and rigors which used to follow salvarsan administration are now avoided by the simple precaution of boiling the distilled water in which the drug is dissolved a short time before use. The blood should be tested every six months for two years, and further courses of injections given if the Wassermann reaction prove positive. Certain authorities recommend mercury to be continued for two years even when salvarsan substitutes are employed. MacDonagh has introduced intramine and ferrivine, which he considers to be safer than arsenic compounds. Apart from these new drugs the most effectual remedy in the earlier stages is mercury, which should be taken continuously, whether symptoms be present or not, for at least two years, and subsequently, if symptoms are still present, until at least three months after all evidences of the disease have ceased. It may be given by mouth, by inunction or fumigation, and by injections into the muscles. The precautions to be observed in all cases, in addition to the duration of the treatment just mentioned, are : (i.) The mercury must be gradually increased until the gums become tender, then regulated until toleration is established. It is necessary to get rid of decayed teeth at the very outset, and a mouth-wash of potassium chlorate and weak carbolic acid may be necessary. (ii.) The patient should be seen once or twice a week, so as to watch for salivation, diarrhœa, gastric disturbances, and to regulate the treatment. If the

mercury be temporarily stopped, 5 grains of iodide of potassium should be given thrice daily. Debilitated subjects and subjects of renal or visceral disease require smaller doses and extra caution. (iii.) The patient should absolutely avoid alcohol and tobacco while under treatment, and take plenty of milk and light food. The open-air treatment as for tuberculosis, is given at Aix-la-Chapelle. Sea air and sea voyages are specially beneficial.

Mercury is ordinarily administered by the mouth, liquor hydrargyri perchlor. (with or without potassium iodide) being given thrice daily after meals, gradually increased ; or hyd. c. cret. gr. i. or ii. (0·06-0·12) or pil. hydrargyri gr. i. or ii. (0·06-0·12) may be given with opium, gr. $\frac{1}{10}$ (0·006), twice daily. In private practice a convenient way to give mercury is by means of a pill of hydrargyri iodidi viridi, gr. $\frac{1}{2}$ to 1 (0·03-0·06), with opii, gr. $\frac{1}{4}$ to $\frac{1}{3}$ (0·016-0·02), twice daily. Sarsaparilla and guaiacum, when added to medicines containing mercury or iodide, appear to increase their effects in some cases. The inunction of ung. hydrargyri or hydrarg. oleat. is another method ; a piece the size of a Barcelona nut, diluted with an equal amount of lanoline, should be well rubbed into the abdomen or limbs every night. Inunction, if properly performed, is one of the best methods of treatment ; but it is rarely carried out efficiently in this country. In infancy the favourite methods are the administration of hyd. c. cret., gr. i. or ii. (0·06-0·12), once or twice daily, or the wearing of a broad flannel bandage on which ung. hydrargyri or mercury oleate is spread daily. For adults a cleanly method is the mercurial vapour-bath (F. 4) given daily at first.

The intramuscular injection of mercury—which is clean, convenient, and does not upset the stomach—dates from the time of John Hunter, and has come into vogue again during the last few years, many different preparations having been used. I have been well content with a solution of perchloride of mercury (F. 24), to which 1 or 2 per cent. of cocaine may be added to prevent pain. A sterilisable hypodermic syringe is used, the needle being made of platino-iridium (which resists the action of the mercury) and being of rather larger bore than ordinary hypodermic needles. The skin and the hypodermic needle having been rendered aseptic, the solution is injected into the substance of a muscle, preferably the muscle of the buttock. Start with a small dose, about gr. $\frac{1}{10}$ to $\frac{1}{5}$ (0·006-0·012) daily or every other day, until the gums become affected ; gradually increase the dose, and then administer about $\frac{1}{4}$ grain (0·016) once or twice a week, or every second week, according to the state of the gums. Some prefer the succinimide (F. 24), peptonate, cyanate, cyanide or other soluble salt of mercury, and some recommend a solution of biniodide in oil. The insoluble preparations—calomel, metallic mercury, or salicylate of mercury suspended or emulsified in paraffin and injected by means of a special syringe—have been largely used in France and Germany, but I have found the soluble salts to be more convenient, more rapid in their action, and more exact in their dosage. L'huile grise

(mercury suspended in oil) is sometimes cumulative in its effects. Syphilis has been treated by the injection of serum derived from a cantharides plaster from patients with tertiary symptoms; 10 to 40 c.cm. were injected every third or fourth day for one or two months. Refractory cases are said to have improved.

It is in the later *secondary* and in the *tertiary stages* of syphilis and particularly in the internal manifestations, that potassium iodide finds its chief use, and in a large proportion of these cases this alone will promote absorption, if given in large enough doses. My belief is that the doses generally given are not large enough, because I have known many cases, which fail with 5 or 10 grains (0·3,-0·6), succeed with larger doses. My practice is to start with 20 grains (1·2), and rapidly increase the dose to 60 grains (4 gm.) thrice daily after meals, followed by a glass of milk. If the patient has running from the nose and eyes, it may often be relieved by doubling the dose. The largest dose I have given was 90 grains (6 gm.) thrice daily. Iodide spots may sometimes be relieved by 3 drops of liquor arsenicalis, and as the iodide lowers the blood-pressure, spiritus ammoniæ aromat. or other stimulants may be called for. If iodide of potassium disagrees, even when followed by milk, substitute the sodium and ammonium salts. After the symptoms have disappeared the doses may be lowered, but on no account should the iodide be stopped until at least three months later. Mercury may with advantage be added in bad cases or if an insufficiency of the metal has been taken. Bismuth preparations have recently been found to be most useful.

§ 435. IV. **Plumbism** (Synonyms : Saturnism, Chronic Lead Poisoning). Chronic ill-health, usually associated with a number of other symptoms, results from the slow absorption of lead into the system.

Symptoms.—(1) Pallor is very marked, but is not commonly associated with impoverishment of the blood. In acuter cases there may be true anæmia. The pale, pasty appearance of lead workers is well known. Depending as it does on vascular changes, it naturally tails to yield to iron. (2) The gums usually show the so-called " blue line " except in those who clean their teeth regularly. This is a livid line on the gums close to the teeth due to the formation of lead sulphide. In copper poisoning the line is brighter and more greenish. A few medicinal doses of the metal may produce the blue line and also basophil granulation of red blood-cells. (3) Obstinate constipation is usual, and is sooner or later associated with severe colic. The fact that the colic is recurrent is of aid in diagnosis. An acute attack of colic may be associated with a rise of temperature, quick pulse and general malaise with acute abdominal pain and some degree of tenderness and rigidity. (4) Lead is found in the urine in some cases, but it is to be borne in mind that the effects of lead poisoning, especially arterial changes, long outlast the actual presence of lead. Slight albuminuria is common. (5) The arterial changes are amongst the most important, leading to chronic headache with high tension pulse, Bright's disease, fainting fits from spasm of

cerebral vessels, or even definite transient hemiplegias. There are also attacks of acute pain resembling the lightning pains of tabes. (6) Lead has a. special tendency to attack the peripheral motor nerves. The musculo-spiral nerve is most commonly affected, leading to wrist drop. The supinator longus usually escapes, and thus supination is preserved. The muscles waste rapidly. Almost all the recognised types of muscular paralysis have been noted, but these are not so typical of chronic as of the more acute cases. Sometimes the paralysis is generalised. Tremor is often present in cases without paralysis, more especially if the lead has entered the system by inhalation, as in glass-blowers. Optic neuritis and various degrees of amblyopia may occur. A neurasthenic condition is common, and the mental·condition may be such that the patient is unfit to look after himself. Acute mania, convulsions, etc. (known as saturnine encephalopathy), are sometimes met with.

Diagnosis.—Early arterial changes in a lead worker, especially if accompanied by a blue line or paralysis, or by constipation and recurring colic, are diagnostic. It must be noted that symptoms may not super-vene while the patient is working in lead, but only occur when he leaves this for a more active employment, which has the effect of " flooding " the system with a relatively large dose of lead. This has been especially noticeable among workmen just become soldiers.

Prognosis.—This is good only in early cases who can avoid further exposure to lead. In others paralysis may clear up, but other symptoms will follow. The disease does not greatly shorten life.

Etiology.—(1) Painters, lead workers, plumbers, pewterers, glaziers, file cutters, glass workers, and many others are exposed to lead in their occupations. In most cases the lead is taken direct into the mouth with food. In others it enters the lungs by inhalation. (2) The disease may occur in epidemics owing to the consumption of contaminated water which has been stored in leaden cisterns, or has passed through leaden pipes. A specially soft water is apt to dissolve a certain amount of lead carbonate usually found inside leaden pipes. In other epidemics, such as that of Newcastle in 1900, the water has been found to be slightly acid near its source. (3) Various articles of food or drink are apt to become con-taminated when stored in lead, lead-glazed, or pewter vessels, such as tinned provisions, beer, cider, or wines. Cases still occur in men who have drunk the beer first drawn off in the morning which has lain some hours in a pewter or leaden pipe. (4) Rarer cases are the sleeping in newly-painted rooms, and occasionally in susceptible persons the external applica-tion of lead lotions and ·powders may produce poisoning. The internal administration of lead as an abortifacient is still practised in some Midland towns. Any age or sex may be affected, but women are more susceptible than men. A first attack may not occur until after exposure for many years. Alcohol predisposes.

Treatment.—The first indication is the avoidance of the cause, and those who are exposed to the poison by reason of their occupation should

observe the greatest personal cleanliness. The face, hands, and teeth should be cleansed before meals. The ventilation of the workroom should be supervised, and a respirator worn if the air contain much dust. Sir Thomas Oliver has a poor opinion of the prophylactic value of sulphuric acid lemonade, inasmuch as the sulphate of lead is hardly less soluble than the carbonate. Fruits and alcohols should be avoided. Saline aperients and small doses of iodide of potassium should be administered, but the latter should be increased with caution, so as to avoid flooding the blood with a soluble lead salt. Colic is best treated by codeine gr. ½–1 combined with liquid paraffin as a laxative. The treatment of the constipation, colic, paralytic and other nervous lesions will be found in their respective sections. For the removal of lead from the system, Sir Thomas Oliver urges the use of the arm and leg electrical bath daily; the negative pole in the arm, the positive in the foot bath. A current of 20 to 40 m.a. is run through for half an hour. In all acute cases rest in bed is essential.

V. **Incipient Tuberculosis** is generally attended by anæmia, pallor, weakness, and loss of flesh (§ 110). The anæmia is often very marked, and if a young anæmic patient is not amenable to treatment by iron, latent tuberculosis should always be suspected (Trousseau). The disease may be entirely latent in the sense of being unattended by any physical signs in the lungs or elsewhere. It is useful to remember that a tuberculous process, no matter where it is situated, is always attended by pyrexia of an intermittent type, though this is apt to be overlooked. Inquiry should therefore be made for sweatings or " chills," careful temperature readings should be procured, and the sputum and urine should be examined for the bacillus. The early diagnosis of tubercle in the lungs, meninges, kidneys, peritoneum, and other parts has been given in their appropriate places. Early spinal caries may also be overlooked, and the only symptoms present may be slight pain in the hypogastrium.

VI. Incipient or **Latent Carcinoma** and sarcoma are also attended by pallor, weakness, and emaciation; they form the essential parts of cancerous cachexia. The pallor does not yield to iron. Emaciation is, however, usually the most constant and most prominent feature, and therefore malignant disease will be considered fully under that symptom (§ 445). I have twice mistaken cases of scirrhus of the pylorus for examples of primary anæmia. They were cases in which the pyloric end of the stomach was drawn up under the liver, and therefore local signs of the disease entirely escaped detection during life. The diagnosis is all the more difficult when it occurs, as it did in these cases, without vomiting, and in comparatively young women, aged twenty-eight and thirty-five respectively. There may be hæmic murmurs, and on rare occasions intermitting pyrexia. Nowadays an expert opinion on the blood would almost certainly prevent such an error. Another case of anæmia which came under my notice for a severe persistent neuralgia of the third sacral nerve, lasting many months, eventually proved to be carcinoma of the

prostate. Repeated careful examination should be made of all the abdominal and pelvic organs and of the blood.

VII. **Dyspepsia, Constipation, Colitis,** and various other disorders of the **alimentary canal** frequently come under our notice for pallor. Indeed, dyspepsia and confinement indoors are perhaps the commonest causes of pallor among hospital out-patients, and it must not be forgotten that apical abscesses and pyorrhœa alveolaris may be a potent cause of intense anæmia. Deficient food, and particularly deficient nitrogenous food, may also act in a lesser degree. In dyspeptics the'pallor is of a peculiar kind, in which the skin loses its lustre and may even be scurfy, and there are dark rings under the eyes. In colitis and other intestinal affections the same may be observed, and it is wonderful what an improvement in the patient's appearance is made after a course of treatment by salicylate of bismuth, naphthol, or other intestinal antiseptics, combined with purgatives. Many cases in this group are autotoxæmias.

VIII. **Aortic Valvular Disease** often presents a sallowness which may be mistaken for the pallor of primary anæmia. The patient is usually an adult at or past middle life. In aortic regurgitation the cardiac murmur and the pulse (§ 53) are sufficiently characteristic for the detection of the disease on examination. But in aortic obstruction the most experienced auscultators may fail to detect or may misinterpret the signs of the lesion.

IX. **Chronic Renal Disease** is sometimes accompanied by a pallor which may readily be mistaken for primary anæmia. This is especially the case in chronic parenchymatous nephritis, which is apt to affect young people. The pallor, however, is of an ivory whiteness, is usually accompanied by a certain amount of dropsy, and the urine reveals a definite amount of albumen and tube casts. Chronic interstitial nephritis is usually met with in older people ; it is generally attended by sallowness, but is mentioned below under progressive asthenia, which is its more constant and striking symptom.

X. **Cirrhosis of the Liver** may be attended by an anæmic pallor ; but it is usually attended also by dilatation of the venous capillaries in the face, which are very characteristic. The signs and symptoms of this disease may be very obscure.

*Certain other secondary anæmias are readily detected by their history—*viz., HÆMOR-RHAGE, or long-continued drain on the system ; CHRONIC SUPPURATION, or other septic processes ; RHEUMATIC and other FEBRILE CONDITIONS.

XI. Anæmia may be due to hæmorrhage—profuse and sudden, or small and frequent ; hyperlactation, profuse chronic diarrhœa, and other debilitating conditions. Such cases are generally discoverable by a history of the cause, such as menorrhagia, post-partum or ante-partum hæmorrhage, bleeding-piles, melæna, recurrent epistaxis, hæmatemesis, hæmoptysis, hæmaturia, etc. The treatment resolves itself into attacking the cause. But in severe cases of post-hæmorrhagic and other forms of intense anæmia, and whenever collapse is present, transfusion or infusion may be called for.

§ 436. INFUSION OF SALINE SOLUTION may be indicated in the presence of shock, collapse, or intense anæmia, arising in three groups of conditions : (1) Acute hæmorrhage, resulting from abortion, post-partum, and other causes of uterine hæmorrhage, gastric or intestinal ulceration, operations attended by profuse bleeding, or internal

hæmorrhage after abdominal injuries.   (2) Collapse consèquent upon surgical opera-
tions, cholera, or severe diarrhœa due to any cause.   (3) Blood poisoning—*e.g.*, in
uræmia, puerperal eclampsia, diabetic coma, or poisoning by carbolic acid, strychnine,
or phosphorus.   Infusion of saline solution can be rapidly and easily performed, and
skilled assistance is not necessary.   As a mere diluent or to make up the volume
of the blood it is of great value.   In cases of blood poisoning (Group 3 above) vene-
section should be performed to eliminate the poison before giving the saline.   Sodium
chloride (a neutral salt) is used to bring the specific gravity of the fluid to be injected
up to that of the blood plasma.   The normal saline solution (0·75 per cent.) is pre-
pared very readily by dissolving 1 drachm (or to be exact 66 grains) of common
salt in a pint of *boiled* water.   Hypertonic saline solutions are giving good results in
cholera (§ 245).   The infusion may be made (1) into a vein ; (2) into the subcutaneous
cellular tissue of the thighs, anterior abdominal wall, below the clavicle, or mammæ ;
or (3) into the rectum.   An ordinary exploring needle connected to a rubber tube
5 to 7 feet long, with a glass funnel or a douche-can—all rendered aseptic—and a
thermometer, complete the necessary apparatus.   The needle is inserted after cleans-
ing the skin, and the operator standing on a chair pours the solution (at a temperature
of 99° or 100° F.) from a height of 3 to 6 feet above the needle slowly into the funnel.
Three to six ounces may be injected at intervals of ten to fifteen minutes.   If neces-
sary, more than one puncture may be made.   Usually ⅓ to ¾ pint is sufficient, but 2
to 3 pints may be needed.   The puncture must afterwards be sealed with collodion.
The danger of abscess or sloughing is very slight if the infusion be given slowly ; not
too much in one place, and with thorough asepsis.   To inject saline solution into the
rectum a similar apparatus with a rectal tube is employed.   The douche-can should
be held only a foot above the level of the patient, and the tube clamped so that the
fluid can be delivered in very small quantity.   This method is simple, yet often of
great service, particularly in the collapse after operations.

TRANSFUSION OF BLOOD may be called for in a variety of conditions, but is most
commonly employed in cases of shock and of severe anæmia.   Where these are com-
bined it is especially useful.   The procedure is as follows :   A suitable donor must
first be obtained.   Any blood is suitable which neither agglutinates the patient's
red cells nor is itself agglutinated by the patient's serum.   A rough but quite
sufficient test may be made as follows: A drop of the patient's blood is mixed
with a drop from the prospective donor on an ordinary glass slide.   A cover-slip
placed over it may with advantage be ringed with vaseline.   If, after five
minutes, there is no agglutination of red cells visible under the microscope, the
donor is suitable.   Otherwise another donor must be chosen.   More elaborate methods
are only applicable where already tested donors are available, as is the case in some
hospitals.   The test must be repeated before every transfusion, even if the same donor
be employed.

Everything must be in readiness before any blood is drawn.   The donor is prepared
by being placed in a recumbent position, both arms being bared to the shoulder.   A
sphygmomanometer affords the most convenient method of compressing the veins ;
where it is not available, a bandage may be used.   The other arm is in reserve and
will usually not be required.   Both arms are cleaned with soap and water and an
antiseptic, but iodine must not be used as it obscures the veins.   The patient is
similarly prepared.   The operation consists in withdrawing blood by means of a
needle from the donor, passing it into citrate solution at blood heat and immediately
re-injecting it into the patient through another needle.   The following apparatus is
therefore necessary: (1) A 2 per cent. solution of sodium citrate in 0·8 per cent.
saline is placed in a clean flask or bottle and kept up to blood heat in a basin of hot
water.   An assistant must be instructed to see that the water is kept hot.   About
2 ounces will be required for each 10 ounces of blood, but the exact amount is not
of great importance.   (2) A short sharp needle with a rather flat bevel (lest the veins
be pierced) and rubber tubing.   The permeability of both must be tested.   The
needle must be clean and smooth inside as well as out.   (3) A second needle and tubing

and a large syringe for injecting the citrated blood. A funnel and tubing may be employed, but are more difficult to keep warm. If used they must of course be immersed in hot saline, and only saline or citrate saline is to be used for washing needles, etc. (4) A suitable cannula, ligatures, scissors, scalpel, etc., should be at hand in case the operator is not successful in entering a vein, as may happen if he has little experience.

When everything is ready the sphygmomanometer is tightened round the donor's arm so as to constrict the veins. He is told to open and close his fist several times which will congest the veins and make them stand out. A needle is thrust into the most prominent vein and the citrate bottle filled with blood—usually ten to twenty ounces are taken. The citrate bottle must be kept warm and gently agitated to ensure mixture of the citrate and blood. The sphygmomanometer is released and the needle withdrawn in this order. A pad and bandage are then applied and the donor should usually lie quiet for an hour or so. The arm of the patient is then constricted in order to throw up a vein, but as soon as the needle enters the vein, the constriction is removed and the citrated blood injected or run in. In a proportion of cases a certain amount of discomfort is felt and the patient may experience a slight rigor. If the rigor is severe, or any signs of collapse appear, the transfusion must be stopped, but these are unusual if there has been no time lost and the preliminary testing has been accurate. Where special apparatus or skill are available improved methods of transfusion can be used. The above method is within the reach of every one. It is desirable that the saline-citrate solution, or tablets for making it, should be kept ready at hospitals and even by the practitioner. A small transfusion may be a matter of urgency; for example, in purpura and in hæmophilia neonatorum. In such cases it will suffice if a large serum syringe be one-fifth filled with citrate saline, then filled with blood by venipuncture, and forthwith re-injected into the superior longitudinal sinus or beneath the skin of the child.

XII. **Chronic Suppuration** (with or without amyloid disease) and other septic processes and causes of a prolonged drain on the albuminous materials of the blood are potent sources of anæmia. Prolonged lactation may also act in this way.

XIII. **Post-febrile Anæmia,** associated nearly always with weakness and emaciation, may ensue after rheumatic fever, enteric fever, varicella, malaria, and indeed after any of the acute specific fevers.

XIV. Finally, in various conditions referred to in Group II., p. 631 (Emaciation), or Group III. (Debility), Pallor may be the symptom which first attracts our notice, for these three important symptoms are so often associated. Early myxœdema (§ 449) is one of these, and the puffiness of the eyes, the failing memory, loss of hair, and bodily weakness may for a time escape observation, or be attributed to other causes. Myelopathic albumosuria also may first come under notice for anæmia.

Rarer Causes of Anæmia.

The patient is **pale** ; *there is* ENLARGEMENT OF THE SPLEEN, *or the* LYMPHATIC GLANDS, *or both, and* EXAMINATION OF THE BLOOD *may reveal characteristic changes.* The disease is probably LEUKÆMIA, HODGKIN'S DISEASE, SPLENIC ANÆMIA, CHOLÆMIA, or a SEQUELA of MALARIA.

§ 437. I. **Leukæmia** or **Leucocythæmia** is a comparatively rare disease characterised by progressive anæmia, a large and persistent increase of white corpuscles, a slight diminution of the red cells, accompanied by enlargement of the spleen. There are two varieties of the disease : (a) *Myelæmia*, the spleno-medullary type, and (b) *lymphæmia*, the lymphatic type. The former is due to an apparently purposeless overgrowth of the bone-marrow, the latter to a similar activity of the lymphatic tissues. In the first

variety the circulating blood is found to contain excess of cells of myeloid origin ; in the latter an excess of lymphocytes. In many of their clinical manifestations they are very similar. Either variety may occur as a chronic or as an acute disease.

MYELÆMIA.—*Symptoms.*—These are often indefinite in the early stages. The patient may only complain of general weakness and debility and the pallor may not be very marked until late in the disease. In other cases the symptoms first complained of may be epistaxis, vague pains, dyspnœa, enlargement of the abdomen, or cerebral symptoms. As the disease develops the symptoms are seen to fall into two groups— those due to the blood condition and the resulting toxæmia, and pressure symptoms due to the presence of nodules of new growth in various situations. In the first group the most important are cachexia, weakness, and hæmorrhages. Pyrexia of an irregular type is present in three-fourths of the cases. The urine shows an excess of uric acid. In the second group come enlargement of the spleen, and pain and discomfort due to this or to perisplenitis ; slight or sometimes enormous enlargement of the liver, enlarge- ment of lymphatic glands, infiltration of the skin, gastro-intestinal disturbance, dysphagia, dyspnœa, and ascites. The splenic enlargement may be enormous, ex- tending into the pelvis ; sometimes the patient comes first complaining of abdominal swelling. *Acute* spleno-medullary leukæmia resembles acute lymphatic leukæmia (see below) in its clinical course. It is especially apt to be associated with numerous tumours in various parts of the body, due to the lighting up into new activity of rem- nants of the marrow tissues of the fœtus. The blood cells are usually more primitive than is the case in the chronic form, and may with difficulty be differentiated from " large lymphocytes." There are, however, a few eosinophil myelocytes, which are not found in the lymphatic form.

Etiology.—The acute form of the disease is more frequent in younger persons, but, generally speaking, myelæmia is a disease of adults, from twenty to fifty years of age. It affects men more often than women. There is reason to suppose that the disease is infective, but nothing definite is known as to its cause.

Diagnosis.—The most striking point is enlargement of the spleen ; this will often suggest the presence of the disease, but the diagnosis ultimately rests on the con- dition of the blood, in which are found a large excess of all cells formed in the marrow —*i.e.*, (i.) myelocytes of all sorts ; (ii.) nucleated red cells, and an increase also of (iii.) the leucocytes normally found in the blood-stream—*i.e.*, the polymorphonuclears, mast cells and eosinophils. The leucocytosis varies between 100,000 and 500,000 or more in average cases ; the latter figure is quite usual. Towards the close of the disease the red cells undergo marked diminution.

Prognosis.—This, in the last resort, is serious, as very few cases have been known to recover. Remissions in which the patient regains health and the blood becomes normal may last for two or three years, but are usually followed by recurrence. These remissions can, as a rule, be brought about by the use of X-rays. The disease is essentially chronic, and may last ten years ; it seldom lasts less than one year. Death may result from asthenia, or from complications such as cerebral hæmorrhage, other hæmorrhages with much loss of blood, or severe diarrhœa.

Treatment.—Arsenic is the only drug which seems to have at all a specific action. It should be given in progressive doses, and when a remission has been brought about the patient should continue to take the drug as long as the blood shows any abnor- mality. Unfortunately, arsenic seems to lose its power after a time. The X-ray treatment of this disease (filtered pastille doses every seventh or tenth day) gives good results. It is essential that the patient should submit to periodical blood examina- tions in order that X-ray treatment may be commenced again as soon as the blood shows signs of a recurrence. Benzol also may be tried, as described below. Splenec- tomy is not indicated, although it has produced temporary benefit.

LYMPHÆMIA usually occurs in an acute form. The *Symptoms* may be very various when the patient first comes under treatment, a frequent one being stomatitis, due to the breaking down of small lymphoid nodules beneath the epithelium of the gums, with subsequent infection and hæmorrhage. (i.) Rapidly progressive anæmia with

asthenia and hæmorrhages ; (ii.) enlargement of the spleen, liver, kidneys, and lymphatic glands. (iii.) Tumours similar in distribution to those seen in spleno-medullary leukæmia are more common in this form of leukæmia and also more frequently associated with chloromatous changes. (iv.) The blood shows excess of lymphocytes, which vary considerably in their form and staining reactions (Plate III.). The average number is about 50,000 to 200,000 per c.mm. There are usually only a few nucleated red cells to be found, and mitotic figures are not uncommon, although perhaps less constant than in the spleno-medullary form. *Chronic lymphatic leukæmia* resembles Hodgkin's disease in most of its features ; often the blood condition is the only feature which differentiates the two. As a rule all groups of the lymphatic glands enlarge at the same time, whereas in Hodgkin's disease the enlargement of one group usually precedes that of any other. Lymphoid growths in the walls of the stomach may lead to dyspepsia and vomiting.

TABLE XXV.- DIFFERENTIAL DIAGNOSIS OF THE LEUKÆMIAS,
SPLENIC ANÆMIA, ETC.

	Enlargement of Spleen.	Enlargement of Glands.	Leucocytosis.	Other Characteristics.
Myelæmia (*Spleno-medullary Leukæmia*).	Massive.	Usually none.	100,000 to 500,000, with myelocytosis	Progressive anæmia ; liver often enlarged.
Lymphæmia (*Lymphatic Leukæmia*).	Usually slight ; rarely massive.	Moderate, soft as a rule.	50,000 to 200,000, with lymphocytosis.	Progressive anæmia.
Hodgkin's Disease.	Slight ; less commonly massive.	Marked, and hard as a rule.	None or slight.	Little anæmia until late stages.
Splenic Anæmia.	Massive.	None.	Leucopenia.	Gastric hæmorrhage ; cirrhosis of liver later.
Gaucher Splenomegaly.	Massive.	None.	Leucopenia.	Familial pigmentation of face, hands, sclera ; red cells not fragile.
Acholuric Jaundice.	Massive.	None.	None or slight.	Familial ; jaundice ; red cells unduly fragile.

Etiology.—Males are more frequently affected than females. Children are attacked with relative frequency, but the disease has been seen at the age of seventy-three.

The *Diagnosis* depends on the finding of the characteristic blood changes.

Prognosis.—The disease is usually acute, lasting not more than six or eight months, and it may be much more rapid. Complications are as in the spleno-medullary form. Thrombosis is a rare complication, affecting especially the corpora cavernosa. The chronic form may last two to three years or as many as ten.

Treatment.—This is in the main symptomatic. Arsenic is indicated, as in the spleno-medullary variety of the disease. Benzol and olive oil (āā ℥ xv. (1), in capsule, in gradually increasing doses) has given good results in some cases when other treatment failed. A marked or continuous fall of the red cells, or symptoms of poisoning (giddiness and gastric symptoms) contra-indicate its continuance. Great care should be taken not to cause any abrasion of the skin, as the healing power is very defective. X-ray applications should be tried.

Chloroma is a term applied to green leukæmic growths met with in the periosteum of the head and face, and in other parts where leukæmic growths may occur. It is a rare manifestation of leukæmia, somewhat similar to lymphosarcoma in its clinical characters. The green colour may be obvious in the urine.

§ 438. II. **Hodgkin's Disease** (Synonym : Lymphadenoma) is a disease characterised by anæmia, progressive hyperplasia of the lymphatic glands, and sometimes lymphoid growths in the liver, spleen, kidney, and other organs. There are two forms of the glandular enlargement, soft and hard. In the former the glands are soft in

consistence and somewhat enlarged ; there is a proliferation of the endothelial cells, dilatation of blood-vessels and lymph sinuses, while masses of lymphocytes crowd the lymph sinuses, and large multinuclear cells also occur.   In the hard variety, which is usually a more advanced form, the glands are much enlarged and hard in consistence, and there is a great increase in the fibrous tissue which takes place at the expense of the other cells.

Symptoms.—(1) Sometimes debility and anæmia are the first symptoms, but more frequently enlargement of the lymphatic glands of the neck, axillæ, or groins first attract attention.   The enlargement of the cervical or other single group of glands may precede that of any other glands for a considerable time, even for a few years, but usually the extension to other glands is more rapid.   In chronic forms of Hodgkin's disease the glands are hard, separate, and movable under the skin ; but in the acuter forms the glands feel soft.   For accurate diagnosis a gland should be excised under local anæsthesia, and examined microscopically.   In Hodgkin's disease the glands readily become the seat of tuberculous infection ; hence probably the variation in the clinical symptoms and course.   (2) Pressure effects occur when the deep glands—*e.g.*, in the thorax—become involved, and occasionally they are the first to enlarge.   The pressure symptoms are described in § 66.   Bronzing of the skin may arise as a consequence of pressure on the solar plexus.   (3) Irregular paroxysms of intermittent pyrexia occur at intervals of a few days or a few weeks, and these attacks may coincide with a paroxysmal enlargement of the lymphatic glands.   (4) The spleen enlarges as the glands enlarge, but the enlargement is seldom very great ; usually the edge can just be felt below the costal margin.   The liver also enlarges in some cases.   (5) The blood changes are not characteristic ; they consist chiefly of diminution of the hæmoglobin and the number of red corpuscles with poikilocytosis in the later stages.   (6) The constitutional symptoms, anæmia, and languor increase, and in the later stage may become extreme with the concomitant symptoms of emaciation and a marked tendency to hæmorrhage (as is usual in all profound anæmia).   (7) Pruritus may be very severe, especially if there be nodules of growth in the skin.

Diagnosis.—Clinically, Hodgkin's disease and the more chronic cases of the *lymphatic variety of leukæmia* are alike, but an examination of the blood at once reveals the difference.   The blood in Hodgkin's disease shows only a diminution of the hæmoglobin and the red cells, and in the later stages only a slight degree of polynuclear leucocytosis ; in leukæmia there is always a *marked and characteristic increase in the number of lymphocytes* throughout, and usually an increase in the total number of leucocytes.   *Lymphosarcoma* has been considered on the Continent to be akin to Hodgkin's disease.   In lymphosarcoma the growth, though primarily involving the lymphatic glands, invades the surrounding tissue, and thus reveals its malignancy.   The diagnosis from *tuberculous adenitis* is often difficult.   Tubercle is more common at an earlier age ; the glands tend to be matted together, and to caseate or suppurate.   The tuberculin tests, if positive, prove the presence of tubercle, but they cannot decide whether the morbid process in a given case was tuberculous at the onset.   *Syphilitic* glandular enlargement is preceded by the appearance of a chancre ; the glands are very hard, and tend to disappear rather than to spread.

Prognosis.—Hodgkin's disease usually runs a slow chronic course, months, or even years elapsing before extension from one group of glands to another.   On the other hand, the disease may run an acute course, all the glands enlarging within a few months.   Cases are reported to have recovered or improved or remained stationary for a long period without special treatment, and chronic cases have perhaps recovered under treatment.   In severe cases the anæmia and emaciation are marked, and death occurs from exhaustion, or with delirium and coma.   Complications such as pneumonia, pleural effusion, pressure on the bronchi or trachea may also cause death.   Difficulty in swallowing may arise from overgrowth of the adenoid tissue in the pharynx or thorax.

Of the *Etiology* little is known.   The disease usually arises in the first half of life.   Among Sir William Gower's 100 cases 30 were under twenty years old, 34 between

twenty and forty, and 36 over forty. It is three times as frequent in men as in women. An infective origin has been suspected. In some cases the disease has been attributed to an attenuated tuberculous infection. In other cases a local cause of irritation, such as nasal catarrh or a bad tooth, has led to a local enlargement of a group of glands which was followed later by a generalised lymphadenomatous enlargement.

Treatment.—Local chronic groups of glands should be removed. Recoveries have been reported with the use of arsenic. It should be administered for months at a time in gradually increasing doses until the limit of tolerance is reached. Iodides are useless. Phosphorus should be tried if arsenic is ill borne. Tonics, cod-liver oil, and all other means to keep up the patient's strength are useful.

§ 439. III. **Splenic Anæmia** is a rare disease. the characters of which are : (1) Splenic enlargement which cannot be connected with any recognised cause ; (2) absence of any enlargement of the lymphatic glands ; (3) secondary anæmia ; (4) leucopenia, or at most no leucocytosis ; (5) an extremely prolonged course lasting years ; and (6) a tendency to hæmorrhages, especially gastro-intestinal, from time to time. Its nature is still in doubt, but there is an increasing tendency to consider that this term includes a large number of cases of generalised reaction to infection of the lymphoid tissues. The original source of infection may be found in the intestinal tract, the heart, etc. It is undoubtedly true that chronic infective Splenomegalies are much more frequent than has been recognised in the past. The patient may only come under observation during the *second* stage of the disease when anæmia with its concurrent symptoms is complained of. In most cases the splenic enlargement appears to precede the anæmia, and the patient may not seek advice until his spleen has reached the umbilicus. There is great enlargement of the spleen, accompanied by attacks of pain during periods of enlargement of the organ. The spleen in this disease attains an enormous size, often as great as that which occurs in leukæmia. The enlargement is due to fibrosis of the organ, with atrophy of the Malpighian bodies. As the disease progresses there is loss of strength without emaciation, accompanied by gastric disturbance and a tendency to hæmorrhages. Sometimes there is a moderate enlargement of the liver. Pyrexia is present during the active stages of the disease. The blood shows a diminution of the number of the red cells, and a greater diminution of the hæmoglobin. Poikilocytosis may be present. In the *third* stage of the disease all the symptoms are aggravated, and in a few cases of the disease the so-called " Banti's disease " supervenes, with fatal termination. The name " Banti's disease " has been given to a group of symptoms comprising cirrhosis of the liver, jaundice, and ascites, consequent on splenic anæmia, as described above.

The *Diagnosis* from most forms of *secondary anæmia* is effected by the great enlargement of the spleen, and from *leukæmia* by the characteristic blood changes in that disease (§ 437). *Pernicious anæmia* is rarely associated with an enlarged spleen, but difficulty may arise when the blood changes in a severe case of splenic anæmia resemble those of pernicious anæmia. The chief practical difficulty lies in diagnosing the disease from *cirrhosis of the liver* with accompanying enlargement of the spleen. If the red corpuscles and the hæmoglobin rapidly increase under treatment by iron, it is improbable that the condition is due to splenic anæmia. Banti's disease may be almost impossible to diagnose from cirrhosis of the liver unless a history including the blood changes of previous years can be obtained. Some cases regarded during life as splenic anæmia are found after death to be due to *visceral syphilis*, cirrhosis of the liver, or thrombosis of the portal vein. In *Kala-azar* there is a history of residence abroad and liver puncture reveals the parasite.

Prognosis.—The disease is a chronic progressive disorder. It used to be said that death occurred in six months to two years, but it is now known that cases may live ten, twelve or even twenty years after the commencement of the disease. Death takes place by asthenia, occasionally by syncope or hæmorrhage.

Etiology.—Men are more often affected by this disease than women ; it occurs mostly in adult life, but may occur at all ages. The cause is unknown.

The *Treatment* is symptomatic. Arsenic is the most efficacious drug. X-rays may do good. Splenectomy cures, and it is advisable in the early stage; later on, the operative risk is greater.

Gaucher Splenomegaly.—This distinct syndrome has been recognised of recent years. It has the characteristics of splenic anæmia of adults, with certain additional features, namely, (1) pigmentation of a golden [hue, chiefly [on hands, face, and sclerotics; (2) liver enlargement, but no cirrhosis; (3) familial, but not hereditary incidence. Splenectomy is indicated only if performed early, or to relieve symptoms.

IV. In CHOLÆMIA (§ 258) there are anæmia, enlarged spleen, and weakness. It occurs in families, and there are recurrent attacks of jaundice.

The patient is very **pale** *and anæmic, and there are or have been* SORENESS *of the* GUMS, PURPURIC SPOTS, *and brawny indurations of the legs.* The disease is probably SCORBUTUS.   •

§ 440. IV. Scurvy (Synonym : Scorbutus) is a " deficiency disease " due to a too long continued diet in which a certain vitamin is lacking, the so-called water soluble C, which is found chiefly in fresh vegetables. Scurvy is attended by extreme debility, anæmia, sponginess of the gums, and hæmorrhages.

The *Symptoms* start insidiously, and consist of (i.) progressive debility and anæmia, with mental depression and headache, but no pyrexia. Palpitation, hæmic murmurs, syncopal attacks, and other symptoms of anæmia develop. Pains in the back and limbs are usually complained of early. The urine is scanty and highly acid, and may contain albumen. (ii.) The gums become spongy, swollen, and bleed readily. Sloughing may follow, and the teeth become loosened ; the breath is very offensive. Constipation is usual, but diarrhœa with blood may occur later on. (iii.) A characteristic eruption appears, consisting of purpuric spots and swellings of brawny consistence found about the flexures of the joints, especially the popliteal space. These swellings are due to hæmorrhages into or beneath the skin ; if the former, they are purple ; but if beneath the skin, the colour may be pale. Swellings also occur later under the periosteum of the bones of the legs. Epistaxis often occurs, but hæmorrhage from other mucous membranes is not common except in severe cases. (iv.) Death may ensue either from syncope, asthenia, or complications. Among the latter may be mentioned sanguineous effusion into the pleura or meninges, pneumonia, and sloughing of the skin.

Diagnosis.—The diagnosis of scurvy from other causes of *purpuric* eruption is afforded by the condition of the gums, and the hard brawny swellings, which are peculiar to scurvy, and also by the degree of prostration present. Slighter cases are, however, very difficult to diagnose, as similar symptoms may be seen with purpura. Rapid improvement with suitable diet favours a diagnosis of scurvy. A blood count should be made, which would at once distinguish scurvy from the acute blood diseases, which are also accompanied by stomatitis and hæmorrhage. *Syphilitic nodes* on the tibiæ accompanied by cachexia should be carefully differentiated from scurvy, because mercury is so injurious in the latter disease.

Prognosis.—As a rule this is good if the cause be discovered, and removed. Unfavourable symptoms are severe dyspnœa, syncope, scanty urine, and elevations of temperature. Convulsions, hemiplegia, and other cerebral symptoms follow intracranial hæmorrhage. The outlook is grave when dysentery complicates the disease. Necrosis of the jaw or other bones is rare.

Etiology and Treatment.—Scurvy used to be the scourge of the British Navy, until the introduction of lemon juice as a prophylactic. During the recent war there has been abundant opportunity of studying the disease, which till then had become very rare. The research carried on at the Lister Institute by Dr. Harriette Chick and others, established the fact that the antiscorbutic vitamin is contained in large amount in fresh lemons, oranges, and fresh green vegetables ; in moderate amount in roots, such as swedes and potatoes ; and in small amount in milk and

fresh meat. It is absent in dried and preserved foods. As alkalies destroy the vitamin, soda should not be used in cooking vegetables. In expeditions and long voyages, when fresh vegetables are not available, the daily ration should contain an ounce of lemon or orange juice, with sugar, and some peas, beans, or lentils which have germinated. The pulses do not contain the vitamin except when they have germinated. The germinating process is thus carried out. Soak the pulses in water at 60° F. for 24 hours ; drain off the water, spread out in thin layers and keep moist for 48 hours at 60° F. Then cook rapidly (peas 40 to 60, lentils 20 minutes). Prolonged methods of cooking destroy the vitamin ; hence when food containing any vitamin is scarce, roasting and quick boiling is preferable to stewing and simmering. Symptomatic treatment consists in giving a gargle of potassium chlorate or Condy's fluid, and pencilling the gums with a 2 per cent. solution of silver nitrate. Bismuth and opium are needed for diarrhœa, and foods which would irritate such complications as dysentery should be avoided.

The patient is **pale** *and liable to* UNCONTROLLABLE BLEEDINGS, *from little or no cause.* The morbid condition is probably HÆMOPHILIA.

§ 441. V. Hæmophilia is a hereditary disease characterised by a constitutional tendency to uncontrollable hæmorrhage without sufficient cause.

The *Symptoms* are divided into three sets : (1) Hæmorrhages from mucous membranes, or, after some slight injury, from the skin. Nothing abnormal may be noted in a subject of hæmophilia until he has a tooth extracted or a trifling abrasion, when uncontrollable bleeding due to capillary oozing sets in, and lasts for hours or days. When the bleeding occurs from a mucous surface, large blood tumours may form as the blood coagulates. (2) Interstitial hæmorrhages occur spontaneously or after injury in the form of petechiæ or hæmatomata. (3) Affections of the joints, especially the knees and elbows, are met with, and three stages are described : (i.) Recurrent hæmarthroses or effusions of blood into the joints, of acute onset, sometimes attended by pyrexia ; (ii.) reactionary synovitis ; and (iii.) cicatrisation which may lead to permanent deformity.

Diagnosis.—A single severe hæmorrhage does not warrant a diagnosis of hæmophilia, but recurrent hæmorrhages with slight cause are characteristic. The family history of a tendency to bleeding is important. The joint affections are diagnosed by the presence or history of other signs of hæmophilia.

Prognosis.—The disease usually becomes evident during the first few years of life, and as a rule tends to be less troublesome as life advances, disappearing about the age of thirty or forty. Great anæmia occurs from excessive bleeding, and life has been lost after trivial operations such as the extraction of a tooth or circumcision.

Etiology.—Hæmophilia occurs in families for generations. It is met with in males, and very rarely if at all in females, but the diathesis is transmitted through the female who herself may remain unaffected. The cause of the condition is unknown.

Treatment.—Males in a bleeder's family should be guarded from any injury or operation. The daughters are not endangered by parturition, but their sons will probably be bleeders, and their daughters will pass on the same tendency in turn to their offspring. When bleeding occurs, rest is essential, and the best remedy is normal human serum, or horse serum freshly prepared ; failing these, diphtheria antitoxin or a solution of dried serum may be used. Twenty c.c. may be given intravenously, subcutaneously, or applied to the bleeding point.

In **Hæmophilia Calcipriva** the symptoms of hæmophilia occur apart from any hereditary tendency, and are curable by the administration of calcium.

In **Hæmophilia neonatorum** the new-born child begins to bleed about the third day of life from cord, bowel, or elsewhere. Purpura may be present. Injection of serum or whole blood (*e.g.*, from the mother) is specific. Any available serum must be used and 5–10 c.c. should be injected intramuscularly or subcutaneously as early as possible. This may be repeated after twelve hours if required. No other treatment is of any avail.

VI. **Addison's Disease, Morphinism,** and maladies mentioned in Groups II., p. 631, and III., p. 637, occasionally come under our notice for pallor.

There is **pallor** *of the skin and the patient has* BEEN ABROAD. Inquiry should be made for MALARIA, CHRONIC DYSENTERY, WORMS, and other PARASITES, or other TROPICAL DISEASES.

VII. Various tropical diseases and parasitic conditions rarely, if ever, seen in England, are attended by intense anæmia.

(*a*) *Malarial anæmia* is usually accompanied by pigment changes around the eyes and other parts of the body. The history here, of course, is our first clue to diagnosis. The earthy pallor and the enlargement of the spleen (ague-cake) are very characteristic.

(*b*) *Dengue, beri-beri,* and other fevers unknown in this country are accompanied and followed by anæmia, which may be very severe. The diagnosis is made from the signs special to the disease.

(*c*) In Egypt and other countries cases which used to be considered as idiopathic anæmia are now known to be due to *intestinal* and *other parasites,* most of which may be recognised by the presence in the fæces of ova or segments. *Dysentery* may lead to severe anæmia. *Schistosoma hæmatobium,* endemic in Egypt and elsewhere, causes anæmia and hæmaturia (§ 326). The *filaria sanguinis hominis* and *strongylus gigas* also give rise to anæmia.

The **Ankylostoma worm** may be present in the intestine without symptoms, but usually gives rise to anæmia and debility ; exceptionally the case may end fatally. The less serious and earlier symptoms are bronchial catarrh, slight dyspepsia and affections of the skin (papules, pustules and urticaria) due to the passage of the larvæ through it in the process of infection. Melæna is a common symptom. Occasionally there are weakness of body and mind, amblyopia, apathy and melancholia. The two ankylostoma worms, A. duodenale and A. americanum, exist in the adult state in the intestinal tract of man. Their ova are voided with the fæces and undergo further development in them if they are deposited on moist ground. The encapsuled larvæ gain access to the alimentary tract of man again by passing through the skin, then the lungs, up the trachea and down the œsophagus. In their passage they give rise to the symptoms mentioned in skin, lungs, and stomach.

The *Diagnosis* rests on the finding of the characteristic ova in the fæces. The possibility of a helminth infection must be borne in mind in obscure cases of anæmia ; and the two factors which may suggest this are the occurrence of anæmia in epidemic form, especially in European countries among miners, and the discovery of eosinophilia on examining the blood.

The *Prognosis* is good if the patient can be protected from further infection by removal from any district where infection is rife, and especially from work in mines in which the ankylostoma is known to exist. The only adequate method of prevention is by burning the fæces and preventing their deposition in moist places, which favour the development of the embryos.

Treatment.—The best anthelmintic is thymol. Doses of ½ drachm (2) should be given every hour or two hours for three doses, and they will have to be repeated until there are no ova discoverable in the fæces. It is important that the patient while under treatment should not take any alcohol nor fatty nor oily substances ; these may dissolve the thymol and cause poisonous symptoms of absorption. Some patients rapidly improve in health after segregation, without any drug treatment ; this must be ascribed to the absence of reinfection. " Worm-carriers "—those whose fæces contain ova, but who have no symptoms of ankylostomiasis—should be treated, as they are capable of carrying infection.

Tricocephalus Dispar may cause anæmia and enteritis, sometimes a degree of fever, depression and lethargy. The only diagnostic sign is the discovery of the ova in the fæces (§ 239). The eggs mature in moist soil contaminated by the fæces of infected

persons. Treatment is the same as that for ankylostomiasis. Tænia solium and T. mediocanellata rarely cause anæmia. **Oxyuris.**—It is to be remembered that even the common Oxyuris may occasionally cause a fatal anæmia. **Bothriocephalus Latus** is a tapeworm infecting those who eat fish containing the cysticerci. It occurs chiefly in Finland and Central Europe. The patient infected by the bothriocephalus becomes anæmic, sometimes dangerously so. The treatment is similar to that used for tænia solium (§ 239) and anæmia.

Distomiasis.—The earliest symptom is debility, usually combined with some degree of anæmia which increases as the disease progresses, and is one of the chief factors in producing a fatal termination. There is at the same time a dryness of the skin (well known to veterinary surgeons) and general symptoms of intoxication—*e.g.*, fever, malaise, loss of flesh, and slight jaundice. Owing partly to great anæmia and partly to the involvement of the liver, ascites appears. Œdema of the legs and of other parts of the body follows, jaundice deepens, and death occurs by cardiac failure. When the parasite is present in other parts of the body as well as in the liver, the symptoms are more variable, and include abscess of the scalp or of the foot, cavitation of the lungs, etc. Certain symptoms may arise owing to the rupture of the bile ducts in which the parasite is lodged—*e.g.*, general peritonitis or recurring attacks of peri-hepatitis.

Etiology.—The sheep is the usual host of the adult fluke found in this country, the Distoma hepaticum. In the East, infection follows the ingestion of the embryos of an allied parasite, the Distoma sinense. The ova are passed out of the bile ducts of infected animals and so occur in the fæces. If these are deposited near any fresh water, the ovum grows into an embryo, which after certain stages passed in the body of a snail, are conveyed back to man, cattle, etc., in drinking-water or encysted on the leaves of vegetables or grass. From the gastro-intestinal tract they find their way to the bile ducts and may pass through the liver and reach the situations mentioned above. Wherever they settle they give rise to inflammation of the neighbouring tissues. In the liver the resulting fibrosis leads to a spurious cirrhosis of that organ, with consequent ascites, and jaundice.

Prognosis.—In many cases there are no symptoms at all; in others, presumably when the infection is severe or repeated, the train of symptoms above noted appears. There is then little hope of cure as the parasites are placed in a peculiarly inaccessible position. Symptoms of perihepatitis or of the presence of the parasites in the lungs render the prognosis unfavourable.

The *Diagnosis* rests on the discovery of the ova in the fæces or of the parasite (which is about an inch long and half an inch broad) in any part of the body. The presence of parasites may be first suggested by the examination of the blood, which reveals eosinophilia, very marked in some cases.

Treatment is mainly symptomatic, but attempts may be made to wash out the bile ducts with such drugs as euonymin, iridin, or hydrastin, starting with doses of 2 to 3 grains, and increasing if necessary. Other drugs with a cholagogue action prove useful. Urotropine should be tried. All possible measures must be taken to improve the general health.

Distoma Pulmonale (Synonym : Distoma Ringeri) is a parasite found in the lungs, liver, testes, peritoneum, and brain. It is met with in Formosa (being present, according to Manson, in 15 per cent. of the inhabitants), China, and other tropical countries, where it gives rise to endemic hæmoptysis. It measures 8 to 10 mm. in length by 4 to 6 mm. in breadth. It is reddish-brown in colour, oval in form. In the lung, its favourite habitat, it gives rise to chronic cough without physical signs, rusty sputum, and irregular attacks of hæmoptysis, accompanied by intense anæmia. The parasites are coughed up.

§ 442. Anæmia in Children.—All the forms of anæmia above described, with the exception of Chlorosis and Pernicious Anæmia, may occur in children under fourteen, and are produced by the same causes

which affect adults, but they occur in a very different order of frequency and present certain marked differences. (*a*) The spleen tends to become enlarged in all forms of anæmia in children, but is markedly so in (1) splenic anæmia of infancy, (2) myelæmia and lymphæmia, and (3) Hodgkin's disease. (*b*) The blood changes also differ considerably from the blood changes met with in the same diseases in adults. In infancy and childhood slight causes lead to blood alterations of a marked type which, if occurring in an adult, would signify severe disease. (*c*) In secondary anæmias in children, as in adults, a diminution of the hæmoglobin is the earliest change, but there are important differences. In childhood (1) the number of red corpuscles is reduced at a comparatively early stage and new ones enter the blood in a half-formed condition—poikilocytosis and nucleated red cells—and (2) leucocytosis occurs more readily, chiefly of the mononuclear cells (small and large lymphocytes) owing to the activity of the adenoid tissue in children. Adenoid tissue is very active and plentiful in children, and hence in childhood an increase of the lymphocytes (which come from the adenoid tissue and lymphatic glands) is relatively more common than an increase of the polynuclear neutrophil and other white cells (which come from the bone-marrow).

I. PRIMARY ANÆMIAS.—*Pernicious anæmia* is hard to diagnose with certainty in a child ; it is doubtful if it ever occurs. *Chlorosis* in childhood is so rare that some authorities deny its existence. But in countries in which the attainment of puberty is earlier than in Europe, chlorosis also appears at an earlier age. Blood changes with poikilocytosis and nucleated red cells, as met with in the pernicious anæmia of adults, occur in the secondary anæmias of children more readily than in adults.

II. The chief causes of SECONDARY ANÆMIA in children are *defective nutrition*, the *acute specific fevers, acute rheumatism, prolonged suppuration, syphilis, tuberculosis,* and *chronic diarrhœa. Intestinal worms,* and other parasites, such as Bilharzia, may be for long an unsuspected cause of anæmia and debility (§§ 2 and 239). Secondary anæmia frequently occurs in children who have had a deficiency of proteid food. It is met with therefore in children who have been suckled too long, or have had only milk food at an age when they should have had proteid foods containing iron.

In addition to the foregoing, there are three ANÆMIAS SPECIAL TO CHILDREN—III. Infantile Scurvy; IV. Splenic Anæmia Infantum; V. Congenital Anæmia.

§ 443. III. **Infantile Scurvy** (Synonym : Barlow's Disease) used to be considered a variety of rickets, but it is now known that it need not necessarily be associated with rickets.

Symptoms.—(1) The onset may be gradual. The child is noticed to become pale and in late stages profoundly anæmic. Muscular weakness becomes marked, but emaciation may be absent. The child cries when washed or dressed, screams if the legs are touched, and is very still when at rest. (2) Very soon an ill-defined swelling which visibly increases is

seen along the tibia, just above the ankle. The swelling is not necessarily symmetrical, and is due to subperiosteal extravasation of blood. The legs appear as if paralysed, because the child keeps them everted and motionless, dreading the pain caused by movement. Sometimes the femurs are also affected, and there may be œdema of the dorsum of the foot. The arms and scapulæ are next affected ; the ribs, skull, and face rarely so. The joints are free. (3) Swellings may occur in the muscles, resembling abscesses, but there is no redness or fluctuation. (4) Sponginess of the gums develops, and petechiæ and internal hæmorrhages may occur as in adult scurvy. (5) Other symptoms are albuminuria and hæmaturia, and proptosis from hæmorrhage into the orbital periosteum. The *temperature* is normal except after large or recent hæmorrhages, when it may rise to 100° or 102° for a few days.

Diagnosis.—Infantile scurvy may be mistaken for *rheumatism*, but whereas in rheumatism the joints are affected, in scurvy they are free. *Infantile paralysis* is accompanied by no swelling or tenderness of the limbs. With *abscesses, nephritis,* and *stomatitis* there are no signs of scurvy. In *syphilitic* pseudo-paralysis crepitation and pain on moving the limb occurs, due to separation of the cartilage from the diaphysis.

Prognosis. ·· On the whole this is favourable. Under treatment recovery is rapid, and the child may be well in three weeks. If the patient is seen at a late stage, or if from failure to diagnose the disease the diet is not altered, death occurs from syncope or complications such as diarrhœa, bronchitis, and pneumonia, or any of the acute specific fevers.

Etiology.—The disease affects children of six to eighteen months usually, and is due entirely to the absence of fresh food. It is now known to be due to the absence from the dietary of the water-soluble C vitamin. It occurs in infants fed only with proprietary foods, boiled, sterilised, or condensed milk.

Treatment.—The prophylactic treatment consists in the observance of a few simple dietetic rules. Infants should be fed on fresh unboiled milk, or milk which has only been scalded (brought to the boiling-point for a second). Peptonised milk and artificial foods should never be used for longer than a few weeks at a time. Remedial treatment consists in the administration of fresh milk, which contains the vital vitamin. The juice of oranges, lemons, and, in smaller proportion, of swedes, contains the anti-scorbutic food factor. A concentrated form of lemon-juice, from which most of the acidity has been extracted, is now on the market and is of value. For children of a year old, grape, orange, lemon, and swede juice, with potato pulp and raw-meat juice, should be given. The potatoes are steamed, rubbed through a sieve and beaten up with milk to the consistence of thick cream. *Local* treatment consists in wrapping the limb in cotton-wool and preventing movement.

§ 444. IV. **Splenic Anæmia of Children** (Synonyms : Anæmia Infantum Pseudo-leukæmia, von Jaksch's Disease, Anæmia Splenica Infettiva dei Bambini) occurs in children from six months to two years of age, and is characterised by anæmia and

leucocytosis and enlargement of the spleen. The splenic anæmia of adults is not the same disease.

Symptoms.—(i.) Pallor due to anæmia of insidious onset, sometimes preceded, sometimes followed, by (ii.) enlargement of the spleen, which may attain a great size. Attacks of pain may occur, due to perisplenitis. (iii.) The liver is moderately enlarged, and in some cases the glands also. (iv.) There is irregular pyrexia and gastro-intestinal disturbance. (v.) The patient may remain plump throughout, but in severe cases, usually becomes greatly emaciated. (vi.) In serious cases hæmorrhages occur from the mucous membranes and into the skin. (vii.) The blood changes are characteristic —the hæmoglobin is diminished, the number of the red corpuscles is reduced usually to two or three million, and a slight degree of poikilocytosis is present, together with nucleated red corpuscles. Leucocytosis may be absent in the earlier stages, but always marked in the later stages. The polymorphous character of the leucocytosis is a diagnostic feature. Myelocytes, lymphocytes, large mononuclear cells, and many transitional forms are seen, the transitional forms rendering a differential count impossible.

The *Diagnosis* is difficult only in the early stages. In both *syphilis* and *rickets* we often meet with anæmia, enlargement of the spleen and of the liver; but the spleen never attains the same size, and the blood changes are never so marked, as in splenic anæmia of infants. In children severe *secondary anæmia* may present leucocytosis, but the polymorphism of the leucocytes is not found. *Splenic leukæmia* rarely occurs in children, and the leucocytosis has different features. The diagnosis of splenic anæmia of children depends on different features at different stages; and it would appear, from the numerous synonyms above mentioned, that this disease has been described by various observers, under different names, according to the stage under observation. In the early stage the changes in the red corpuscles are prominent, resembling those in severe anæmia. In the later stages the leucocytosis becomes more noticeable; hence the name " pseudo-leukæmia."

The *Prognosis* is good. The course is short, and recovery usually complete, but cases relapse under bad hygienic conditions. Hæmorrhages and petechial eruptions are serious symptoms. The lower the number of red, and the higher the number of white corpuscles the graver is the prognosis. Death occurs from exhaustion or intercurrent diseases.

The *Etiology* is varied; the disease is usually due to some gastro-intestinal toxin. It is often associated with rickets, and sometimes with syphilis.

The *Treatment* consists in remedying the causal conditions. Intestinal disorder must be rectified. Fresh air is essential, and good food, such as yolk of egg, raw-meat juice, potatoes, and bone-marrow should be given. Of drugs, iron and arsenic are the best; cod-liver oil and malt are useful adjuncts.

V. **Congenital Anæmia** occurs occasionally. The causes are obscure. No iron is obtained during the period of suckling, but a child is born with a store of iron in the liver (Bunge), and it may be assumed that this store of iron has for some reason been deficient. Sometimes there is a history of icterus after birth as if there had been an abnormal amount of blood destruction at that time. The condition is very apt to be confused with congenital syphilis. It reacts well to administration of iron.

GROUP II. EMACIATION

WASTING is a common sequence of nearly all acute and many chronic diseases, but when it is the leading or only symptom the following morbid conditions should be borne in mind. The fallacies have been referred to in § 423.

I. Malignant disease.

II. Defective feeding and digestive disorders, colitis, intestinal adhesions or stenosis and other obscure intestinal conditions.

III. Tuberculosis, diabetes mellitus, diabetes insipidus, chronic Bright's disease, syphilis, and other diseases of Groups I and III.

IV. Diseases of the pancreas and other rare conditions.

V. Various nerve conditions.

Marasmus in children may be caused by defective feeding, diarrhœa, constipation, persistent vomiting, hereditary syphilis, rickets, tabes mesenterica, and pulmonary tuberculosis.

Emaciation in the last third of life is suspicious of carcinoma ; in the middle third of life, diseases in II. and III. above ; and in the first third, tuberculosis.

§ **445. I. Malignant Disease** (Carcinoma and Sarcoma) is a cause of emaciation which should be ever present in the mind when the patient is at or past middle age. There are two anatomical varieties— carcinoma and sarcoma. The essential feature common to both is that they tend to recur after removal, to invade the parts around, and to reproduce themselves in distant parts. This is the clinical meaning of the word malignant. Certain localities are much more prone to primary growths than others, and in cases of latent malignant disease it is important to remember the places in which primary carcinoma and sarcoma may occur. The most common positions for *primary carcinoma* are the skin around the mucous orifices, the tongue, œsophagus, stomach, colon, rectum, mamma, uterus and penis. In these regions secondary carcinoma is almost unknown, unless by direct infiltrating invasion. *Sarcomata* are rarely found primarily in these situations, but are prone to start in the glands, the fascia, the bones, the corium, ovary, kidney in children, brain, spinal cord, retina, and in the fibrous structures of the muscles, breast, and testicle. Different varieties vary considerably in their malignancy and rate of growth. The most rapidly growing of all are the large round-celled and the melanotic forms of sarcoma ; the slowest is the fibro-sarcoma. The myeloid sarcoma is of slow growth, and rarely produces secondary tumours. Melanotic sarcoma has a great tendency to reproduction in distant parts. Among the carcinomata the softer or encephaloid varieties are more rapid and malignant than the harder or scirrhous types. Any tissue or organ of the body may be involved either by continuity, or along the lymphatics or by the blood-stream.

Symptoms.—The symptoms of malignant disease necessarily vary with the situation, and under the heading of tumours or growths the diagnosis has been previously dealt with (*e.g.*, the abdomen, § 212, the chest, §§ 66 and 116). We are here concerned with a general review of the symptoms. (1) There is loss of weight quite early in the disease, sometimes long before any local signs can be detected. This is accompanied by a typical cachexia—*i.e.*, an appearance of illness, in which the skin assumes an ashy or sallow hue. The sallowness of the skin may be so marked as to be with difficulty distinguishable from jaundice, or even Addison's disease. (2) The age of the patient is generally advanced in carcinoma, young in sarcoma. The four classical signs of a cancer are pain, swelling, offensive discharge, and hæmorrhage. (3) Pain at the seat of growth is often complained of, especially in varieties which grow rapidly or occur in tense

parts. (4) In accessible situations a thickening, swelling, or tumour may be detected, which is usually hard, nodular, and apt to fix and infiltrate the surrounding parts. Some sarcomata are soft and pulsating. (5) Whenever the growth involves a mucous or epidermal surface there is an offensive pink, sero-sanguineous discharge—*e.g.*, from the vagina. (6) In like manner hæmorrhage may occur, and take the form either of metrorrhagia, coffee-ground vomiting, or melæna ; and when the disease involves the pleura or peritoneum the *effused fluid will be blood-stained*. (7) In carcinoma the neighbouring lymphatic glands become enlarged and palpable. (8) The rate of growth is rapid, though it varies widely in different forms and localities. Scirrhous infiltration of orifices may only reach the thickness of half an inch in six to twelve months, and the patient may live two years ; but a round-celled sarcoma will reach the size of a hen's egg in a month or two and kill in six.

Diagnosis.—Malignant disease may have to be diagnosed from all the other conditions which give rise to emaciation. A malignant nodule may have to be diagnosed from syphilitic gumma (compare, for instance, syphilis of the tongue, skin, etc.), but the latter is usually attended by less pain and constitutional disturbance, and is amenable to anti-syphilitic treatment. Cases of sarcoma are in rare instances attended by pyrexia. It is possible that in the near future a serum diagnosis will be available for the detection of cancer in obscure cases.

The *Prognosis*, if the case is untreated, is always of the gravest kind, the course rarely lasting more than one, or, at the outside, two years. A few cases of undoubted malignant disease have undergone spontaneous involution. The prognosis largely depends upon the stage at which the true nature of the case is detected. On this depends very largely both the prospect of arrest or removal. In general terms the prognosis also depends on (1) the position and accessibility of the growth, how far vital structures are involved, and whether it is on or near the surface ; (2) the structure of the tumour (*vide supra*) ; and (3) the age of the patient, to some extent, for growth is more rapid in the young.

Etiology.—(1) In carcinoma the age of the patient is nearly always over forty, though I have seen cases of scirrhus of the pylorus in persons aged twenty-eight and thirty-three. Sarcoma, on the other hand, may affect children or adults of any age. Sarcoma is the commonest malignant growth of the kidney that is met with under the age of nine. The cause of cancer has so far defied investigation. The recent Cancer Committee summarises the various lines of inquiry and their results. (1) The microbic theory. (2) The embryonic theory, which holds that small islands of cells in fœtal life are isolated and in later life take on independent growth. (3) The theory that cancer cells revert to the type of division peculiar to sex cells. (4) Virchow's view that chronic irritation plays a large part in the causation of cancer. In experimental work the last-named has produced positive results. The irritants known to lead to cancer are (1) intense insolation ; (2) certain animal parasites are often

followed by cancer of the area they infect ; (3) chemical irritants such as gas tar, arsenious acid, unrefined paraffin oil, soot. Malignant growths share the characteristic of young and embryonic tissues of possessing a higher potassium and a lower calcium salt content. Vitamin experiments have led to negative findings. Many observers believe that cancer is in part due to diet ; they point to the fact that the disease is scarcely met with in native races, and that where European and native races live side by side the natives who develop cancer are those who have adopted European diet. Others believe that intestinal stasis is the chief cause of cancer.

Treatment.—Early removal is still the most reliable method. Recently intensive X-ray therapy claims cures in early cases of even deep-seated tumours. Other methods which have succeeded in individual cases are : injection of Coley's mixed toxins ; trypsin and amylopsin, thyroid, Doyen's vaccine, colloidal copper and selenium injections. It is hoped that chemo-therapy may trace something which will have a selective action sufficient to destroy the malignant cells.

II. **Defective Feeding and Digestive Disorders.**—Although malignant disease or tubercle should always be remembered in obscure cases of emaciation in the old and young respectively, perhaps the commonest causes of slight loss of flesh met with in practice are defective feeding and digestive disorders. Digestive disorders may, of course, exist without any wasting, and if the latter be marked and the patient advanced in life it is always suggestive of cancer of the stomach, especially if there be loss of appetite. Defective feeding without digestive troubles, and particularly deficiency in the fats, carbohydrates, and in foods containing vitamins, may without any digestive disorder be attended by emaciation. Defective teeth are a potent source both of digestive troubles and loss of flesh. Various INTESTINAL CONDITIONS are often attended by undue sparseness of body. Among the latent causes of this may be mentioned obscure intestinal stenosis and catarrhal colitis, both of which may be overlooked for a considerable time. Severe diarrhœa is often followed by rapid wasting, especially in children. Incipient cirrhosis of the liver may also be remembered as a cause of emaciation.

III. **Tuberculosis** often first makes itself known to us by an apparently causeless loss of weight. DIABETES MELLITUS and DIABETES INSIPIDUS, CHRONIC BRIGHT'S DISEASE, and other diseases mentioned in Groups I (*ante*) and III (*post*), may first seek medical aid by reason of wasting. This is particularly so in diabetes, where the inconsistency of his ravenous appetite and constant thirst with loss of weight may even impress the patient. On the other hand, some cases of diabetes which occur later in life are associated with a well-nourished if not a full habit of body. Syphilis, so frequently a cause of wasting in infancy, rarely causes much emaciation in the adult, though children affected with the hereditary disease grow up stunted, slight, and delicate. In latent tuberculosis the trunk and limbs may be wasted although the face be plump and rosy.

IV. Among the rarer causes of loss of flesh which should be remembered in obscure cases are obscure visceral disease, and especially disease of the pancreas. Emaciation frequently accompanies kala-azar, beri-beri, and myelopathic albumosuria. .

V. Diseases of the nervous system may sometimes start with or present generalised wasting, such as bulbar paralysis, and the idiopathic myopathies (mostly met with in childhood), but they usually present also their special symptoms.

§ 446. **Marasmus in Children.**—Infants and children emaciate with almost any disorder and with surprising rapidity. A sudden attack of diarrhœa may give rise to loss of flesh in twenty-four hours.

The principal causes are eight in number :

(a) Those which occur chiefly under two years of age : (1) Defective or improper food or feeding ; (2) those associated with diarrhœa or constipation ; (3) those associated with persistent vomiting ; (4) hereditary . syphilis ; and (5) rickets.

(b) Those which are met with chiefly after two years of age : (6) Tuberculous Peritonitis ; (7) Pulmonary Tuberculosis.

(1) DEFECTIVE FEEDING constitutes the commonest cause of emaciation amongst the children of the lower classes. Such children are always fretful, the bowels are irregular and often constipated, the stools, instead of being the normal orange colour of infancy, become either green, grey, or white and " chippy " with particles of undigested food. The error may consist of over-feeding, under-feeding, mal-assimilation or a defect in the quality of the food or the time that it is administered. Over-feeding is perhaps more common than the reverse. Mothers of all classes have a tendency to make their child's feeds too strong and to give them too frequently under the notion that it will make a child " grow strong." The mother's milk, when she is out of health, or when lactation has been too prolonged, may be of too poor a quality to afford adequate nutrition. Preserved milks and foods are lacking in vitamins, and so lead to scurvy (§ 443) and gastro-intestinal disturbance (McCarrison). A good way of giving cod-liver oil (which contains a nutritive vitamin) to children is to pour away the oil from a box of sardines and replace it by cod-liver oil. After standing for twenty-four hours the oil becomes flavoured with the sardines, and both can be given together.

(2) DIARRHŒA or CONSTIPATION, either alone or alternating, are potent causes of wasting in infancy and childhood, and these are frequently due to dietetic errors or want of care and cleanliness in the nursery. The subject of infantile diarrhœa is fully discussed in § 243. Chronic constipation will undoubtedly result in marasmus. In a family with which I was well acquainted the first two children died of marasmus associated with the most obstinate constipation ; the case of the third child, which the mother stated exactly resembled the others in all particulars, was following the same fatal course until systematic treatment by mist. ricini calcis (F. 64) resulted ultimately in restoration to health.

(3) PERSISTENT VOMITING is another cause of wasting in childhood. It may be due to errors of diet, especially too frequent or over-feeding,

or to gastro-intestinal catarrh.   Careful dieting and proper intervals between the feeds will cure most cases.   The reflex and other causes of vomiting (§ 215) must be considered when simple treatment is unavailing.   In intractable cases feeding by the nose has been resorted to.   Hypertrophic stenosis of the pylorus is a rare local cause of vomiting in infants.

(4) HEREDITARY SYPHILIS is a cause of wasting which is generally accompanied by snuffles or skin lesions of some kind (§ 434).   The manifestations of hereditary syphilis always appear during the first year of life, generally during the first six months.   •

(5) RICKETS (§ 478) may be accompanied by wasting, but, as Dr. Judson Bury aptly remarks, " fat rickets are commoner than lean rickets." Rickets may appear at any time between the sixth to the eighteenth month of life ; very rarely after two years of age.

§ 447. (6) **Tuberculous Peritonitis** is a wasting disorder occurring for the most part in children of two years and upwards, due to tuberculosis of the peritoneum and the mesenteric glands.   This form of tuberculous peritonitis was formerly known as tabes mesenterica.

Symptoms.—The onset is very insidious, and may extend over many months. Gradually the limbs and face become shrunken, and there are anæmia, listlessness, vague attacks of pyrexia, and sometimes abdominal cramps.   The leading physical sign is the enlarged abdomen, which is generally tympanitic on percussion.   Sometimes the enlarged glands can be felt, but more frequently there are localised thickenings and masses, which give a doughy feeling, due to chronic tuberculosis of the peritoneum. Attacks of diarrhœa with offensive stools occur from time to time.   If the peritoneum is extensively affected, ascites or matting of intestines is also present (§ 200).   The hectic fever so common in tuberculosis may be present, and sometimes the disease runs a more acute course with pyrexia, resembling typhoid fever, from which it can only be differentiated by the Widal reaction.

Diagnosis.—In addition to the diseases just mentioned, tuberculous peritonitis may have to be distinguished from the distension of the bowels due to improper feeding, in which there is generally no pyrexia, no resistant masses, and disappearance on regulating the diet.   *Rickets* may be attended by a distended abdomen, but has not usually marked emaciation, and the characteristic rachitic changes in the skeleton differentiate it.   In *Hirschsprung's disease* large enemata bring away the masses palpable through the abdominal wall ; and X-ray examination would reveal the distended colon.   In *Syphilitic* cirrhosis of the liver the Wassermann reaction is positive.   *Morbus cœliacus*, a condition occurring in children, in which wasting is accompanied by atonic dilatation of the intestine, and frothy, porridge-like, offensive motions, may be difficult to distinguish, but no glandular masses can be felt.   Tuberculin tests are not reliable.

Prognosis.—The course of the malady is apt to be irregular, with intervals of apparent recovery, followed by relapses.   Sometimes the glands undergo a fibroid change, and what appear to be the most unlikely cases recover.   Among the untoward symptoms are acute local pain and tenderness, indicating peritonitis ; constant diarrhœa, indicating ulceration of the bowels ; and evidences of tubercle elsewhere. The complications are numerous—ulceration of the bowels, attended by pyrexia and intractable diarrhœa ; general tuberculosis ; abscesses forming and bursting in various situations, such as into the peritoneal cavity or from the umbilicus, the latter forming a chronic fistula.   Intestinal obstruction may result at any time from the formation of bands of adhesion.

Etiology.—Tuberculosis of the mesenteric glands may occur at almost any age.   I have met with it at twelve months and also at the age of forty-five, but it is relatively rare under two.   Male children appear to be more prone than females.   The intro-

duction of the tubercle bacillus is the proximate cause, and this gains entrance by the ingestion of milk from tuberculous cows, or milk which has otherwise become contaminated. If the mucous membrane of the alimentary canal is healthy, there seems to be less risk of contamination. The miliary type with ascites and the fibrous adhesive form are both of systemic origin. Other varieties arise by extension from tuberculous enteritis, salpingitis, ileo-cæcal or mesenteric gland tuberculosis.

Treatment.—Prophylactic measures consist in sterilising or Pasteurising the milk, and regulating the supply whence it is obtained. Many cases do well with absolute rest, open-air and properly applied heliotherapy. Dr. A. Rollier's work on this subject must be studied (1923).[1] Uncomplicated cases do well if they pigment well. X-ray treatment does good in the fibrous adhesive types.

GROUP III. DEBILITY ONLY (ASTHENIA)

The causes of debility not necessarily accompanied either by pallor or emaciation are as numerous as those of the two preceding groups, and it must be remembered that all the disorders in both of those groups may commence with weakness only ; in short, the majority of chronic disorders begin with debility. The fallacies (§ 423) and methods of examination have already been given.

Commoner Causes.	Rarer Causes.
I. Senile decay and arterial disease.	I. Myxœdema.
II. Chronic interstitial nephritis.	II. Addison's disease.
III. Neurasthenia, or other incipient or obscure diseases of the nervous system.	III. Bronzed diabetes.
IV. Chronic dyspepsia and obscure diseases within the abdomen.	IV. Disease of the pancreas, Graves' disease, myelopathic albuminosuria, acromegaly, beri-beri, pellagra, and many other conditions mentioned in Groups I and II.
V. Cardiac, tuberculous, and other obscure diseases within the chest.	
VI. Diabetes mellitus and diabetes insipidus.	
VII. Conditions referred to in Groups I and II, in which anæmia or emaciation are ill-marked.	

When a patient is suffering from debility or loss of vigour of mind and body, without any very marked pallor or obvious loss of flesh, and without any marked physical signs or other evidences of disease, in the *first half* of life one would suspect neurasthenia, chronic dyspepsia or gastro-intestinal disorders, incipient or latent tuberculosis, diabetes.

In the *second half* of life one would suspect senile decay, chronic interstitial nephritis, obscure cardiac valvular or aortic disease, diabetes, myxœdema, Addison's disease.

And failing these, some of the conditions previously mentioned among the anæmic or wasting disorders (Groups I and II).

§ 448. I. Senile Decay and Arterial Disease.—As we advance in years

[1] Sir Henry Gauvain gives the following instructions for the sunlight available in this country :-

1st day	..Expose legs to knees for	5 ⎫ minutes hourly
2nd „	.. „ „ „ „ „	10 ⎪ for three
3rd „	.. „ „ „ groins „	10 ⎬ successive hours.
4th „	.. .. Expose legs and buttocks for	20 ⎭
5th „	In addition one aspect of the trunk for five minutes.	
8th „	Total exposure for 20 minutes hourly for three successive hours.	

the power both of body and mind notably declines. This should not be very obvious under sixty, but the age at which it appears differs considerably in different persons, and still more in different families, for the onset of decay in persons, as in plants and animals, is largely a question of heredity plus the previous habits of the individual. Structurally there is a universal tendency to atrophy or degeneration of the parenchyma or functionally active tissues, and slight increase in the lower forms of tissue (such as fibrous and supporting tissues) in all the organs and structures of the body. This is particularly seen in the cardio-vascular system, where the muscular coat (the functionally active tissue of the arteries) first shows signs of senile degeneration.[1]

Symptoms.--Consequent on the changes just mentioned there is a universal lowering of vitality and nutrition, and the general enfeeblement of thought, word, and act which results in the mumbling, fumbling and stumbling of old age. Physical weakness comes on so slowly that even the patient himself is hardly aware of it, and it is not sufficiently recognised that widespread disease of the arteries alone may give rise to progressive mental and bodily enfeeblement at whatever age it comes on.

The following case may be quoted by way of illustration : Jessie T—— was admitted into the Paddington Infirmary in 1889 at the age of forty-nine. At the age of forty-five she began to complain of muscular and mental weakness. This gradually increased, so that at the time of admission she could only walk by pushing a chair before her, and the case was thought, therefore, to be some kind of paraplegia. There were absolutely no physical signs in any organ and no evidences of disease in the nervous system at any time, and the urine was always normal. She became progressively more and more enfeebled in body and mind, gradually took to bed, and died, ten years after admission, of progressive asthenia. I was present at the autopsy in 1899, and all the organs were normal, both macro- and microscopically, with the exception of atrophy ; but there was extreme and widespread disease of all the arteries of the body and of the brain, the main change being granular degeneration of the muscular coat of the heart and arteries, with consequent yielding and great dilatation of the arteries. In infirmary work I have met with arterial disease in persons as young as thirty-eight and forty—cases which were examined post-mortem—and weakness was the only symptom.

The condition of the heart and aorta should be carefully noted, especially any rigidity of the latter, as shown by accentuation of the second sound at the base and the character of a pulse-tracing (Fig. 27). The blood-pressure should be noted from day to day, and the walls of the superficial arteries carefully investigated (§ 76 *et seq.*). Among the later symptoms associated with senile decay of the cardio-vascular and other tissues, perhaps vertigo is the commonest. A large number of other vague cerebral sensations may be experienced, and even convulsions (senile epilepsy) may occur. The urine should always be carefully and repeatedly examined so that senile decay may not be confused with other causes of debility (*infra*), particularly chronic interstitial nephritis.

The *Prognosis* depends upon the amenability of the cardio-vascular

[1] " On Senile Decay," Trans. Med. Soc. of Lond., 1897 ; and " On Arterial Hypermyotrophy and Medial Sclerosis," Trans. Path. Soc. of Lond., 1904.

system to treatment. The diseases to which old age is most liable are of a chronic and degenerative nature, the arterial—*i.e.*, the nutritive system being responsible for this, and itself showing the most definite and wide-spread signs of degeneration. The immediate cause of death in old age is usually some pulmonary complication. An analysis of 409 fatal cases in persons of sixty years of age and upwards, who died consecutively in Paddington Infirmary—February 1, 1886, to December 31, 1892, showed that 121, or 30 per cent., died of some pulmonary condition other than tubercle (pneumonia, bronchitis, hypostatic congestion, and pulmonary apoplexy). The next most fatal disease was cancer, 62 cases (15·5 per cent.), then simple senile decay, 35 cases (9 per cent.), then contracted granular kidney, 24 cases (6 per cent.), then pulmonary tuberculosis, 22 cases (5·5 per cent.).

The *Treatment* should be mainly directed to the cardio-vascular system, and especially to the raising of low blood-pressure and the lowering of high blood-pressure (§§ 72 and 73). Stimulants are nearly always called for in the treatment of disease in the aged. The food should be light, nutritious, and easily assimilable, and small in quantity; it is wonderful how small a quantity of food the aged require, and it has been reckoned that 12 ounces of solid food per diem are sufficient. It is not only useless but harmful to over-feed the aged; keep them warm and prevent chill, but do not over-feed them. Strychnine is *par excellence* the tonic of the aged.

II. **Chronic Interstitial Nephritis** (§ 323) (Chronic Bright's Disease), should always be remembered as a cause of progressive enfeeblement coming on at or past middle life. It is indeed very apt to be mistaken for senility, and failing vigour is the leading symptom for which the patient seeks advice in a large proportion of both these conditions. Sometimes this weakness is accompanied by generalised muscular wasting, but quite as often there is none. The complexion is generally sallow, but there is no definite pallor till late in the disease. Headache is common, chronic interstitial nephritis being one of the commonest causes of headache coming on after middle life.

III. **Neurasthenia** (§ 556) and various other **functional** and **degenerative conditions of the nervous system** may be evidenced by general weakness. This is particularly the case in the functional disorders, such as neurasthenia and hysteria, where the weakness may amount to complete prostration. Such cases are usually met with in the first half of life or middle age. Among the gross lesions which are apt to come on insidiously with weakness are *paralysis agitans*, *bulbar paralysis*, and frontal *cerebral tumour*—diseases more often met with in the second half of life. *Myasthenia gravis* is a rare condition, coming on usually with generalised weakness (see also Generalised Paralyses, § 609).

IV. **Chronic Dyspepsia,** gastric dilatation, and other obscure **diseases within the abdomen** may be attended by debility only for a long time. Gastro-intestinal troubles produce it by chronic toxæmia and mal-assimila-

tion of food. *Mucous colitis* may be specially mentioned in this connection, also *appendicitis, abdominal cancer,* and many of the other conditions mentioned in Chapter IX.

V. Obscure Diseases within the Chest may be manifested by general weakness. *Incipient tuberculosis* of the lungs should always be remembered in cases of unexplained general debility, especially in younger subjects. In the second half of life, and especially in those with an alcoholic history, *cardiac enfeeblement* and fatty heart (§ 62) may cause no other symptom than debility, and the same may be said of *aneurysm* of the third part of the aorta.

VI. Diabetes Mellitus and **Diabetes Insipidus** are often first revealed by progressive weakness, though our attention may also be drawn to these conditions by the thirst, polyuria, emaciation, or glycosuria (§§ 335 and 336). These diseases may occur in either the first or the second half of life.

The **rarer causes of debility** not necessarily accompanied by pallor or emaciation are Myxœdema, Addison's disease, Diseases of the Pancreas, Acromegaly, and some of the conditions mentioned in Groups I (anæmia), and II (emaciation).

§ 449. I. **Myxœdema** (μυξα, mucus ; οιδημα, swelling) is an insidious disease evidenced by weakness, lethargy, and other manifestations of deficiency in the metabolic processes of the body, due to diminished thyroid function. It was so named by the late Dr. W. M. Ord, on account of the mucoid degeneration which takes place in the subcutaneous tissue throughout the body, and is the most obvious anatomical change, producing a kind of solid œdema. It was at first believed by him to constitute a new and hitherto undescribed form of generalised œdema. Marked myxœdema is not a common disease, but the slighter degrees of hypothyroidism are frequently overlooked.

Symptoms.—The weakness here takes the form of a very characteristic slowness of action, of thought, and of speech. It comes on very gradually, and the patient frequently seeks advice for some other reason. (2) The aspect of a marked case is so characteristic that when the doctor has once seen a case he recognises it again directly. The face is slightly puffy, and, the lines of expression being obliterated, it appears immobile and vacant ; the puffiness of the eyelids may be mistaken for dropsy ; the malar capillaries are injected, and cause a characteristic flush on each cheek. The hair of the scalp and eyebrows is scanty and brittle. The speech is slow and drawling, the hands are flat, puffy, and spade-like, and the nails brittle. All the movements are slow, and the mental processes lethargic. (3) As the disease advances, the skin of the whole body is thickened, and at first sight gives the appearance of generalised dropsy ; but myxœdema may be distinguished from the latter by the absence of pitting ; puffy swellings may be noticed above the clavicles. (4) There is a great intolerance of cold ; the skin is dry and scaly ; perspiration never occurs. The temperature is subnormal. (5) The pulse rate is slow and may drop to 40 beats per minute.

Diagnosis.—The disorder may be mistaken in its earlier stages for *anæmia* and the other disorders mentioned in Group I, also for the other causes of *debility* now being considered. A subnormal temperature, and pads over the clavicles, ends of the ribs, or back of the neck, may be the only signs of slight thyroid insufficiency. It may be diagnosed from chronic interstitial nephritis and other forms of chronic renal disease by the absence of pitting on pressure and of the urinary changes of renal disease.

'. *Prognosis.*—Before the introduction of the thyroid treatment advanced cases

tarely lived more than a few years, dying usually of some intercurrent malady. Mental changes, such as hallucinations, even dementia, may supervene. Menorrhagia and other hæmorrhages are sometimes associated with the disease.

Etiology.—The disease is much more frequent in women, in whom it supervenes usually about middle life. It is undoubtedly due to a deficiency of thyroidal function (Athyroidism, § 161). Sometimes a toxæmia, by exhausting the thyroid, leads to hypo-thyroidism.

Treatment.—The treatment by the internal administration of the thyroid gland is so certain and efficacious that this may be used as a means of diagnosis. Tablets of the extract, ¼ to ½ gr. (0·016–0·03), very cautiously increased, may be administered twice or thrice daily after meals. The usual dose of 5 gr. (0·3) is too large to begin with, but eventually some cases can take over 30 grains daily. The drug must never be pushed to the point of producing tachycardia. Complete recovery may ensue after a few weeks' or few months' treatment. Relapses are very apt to occur upon ceasing the thyroid administration and the patient may be obliged to continue treatment indefinitely.

§ 450. II. **Addison's Disease** is a rare malady, described by Dr. Addison [1] in 1854, characterised by progressive loss of strength and general pigmentation of the skin, due to disease of the suprarenal capsules. The most common morbid change in the latter undoubtedly is tuberculosis, which may go on to caseation, fibroid, or other changes.

The *Symptoms* come under five categories : (1) Progressive *general weakness* is its most marked feature and may appear long before any other symptom. It is unaccompanied, as a rule, either by anæmia or marked emaciation until perhaps towards the end. Uncomplicated cases present a subnormal temperature throughout. (2) *Pigmentation* of the skin of a more or less general distribution ensues sooner or later. The colour begins with a yellowish tint, which gradually deepens into a bronze mahogany colour. The localities most affected are the exposed parts (the face, neck, and hands), those where pigmentation is normally present, such as the axillæ and nipples, and sites of pressure (*e.g.*, waist). The edge of a patch of colour shades gradually into the healthy skin around, which makes it difficult to discover such a patch in its early stage. The mucous membranes of the tongue, mouth, and throat frequently present the same kind of patches. (3) *Gastric symptoms* generally occur at some time, such as vomiting, hiccough, and cramp-like pains in the abdomen and loins. Pains in the limbs may also be complained of. The bowels are often constipated, but sometimes there is intractable diarrhœa, which may be fatal. (4) *Cardio-vascular symptoms* may be present—palpitation, dyspnœa, sighing, yawning, and later on a tendency to collapse. (5) *Nervous symptoms* are less common, but may consist of headache, vertigo, and nervousness. The mind is clear, except towards the end, when delirium, convulsions, or coma may set in. These five groups of symptoms vary in their predominance, but asthenia is always present, and pigmentation nearly always. There are two varieties—acute and chronic.

The *Diagnosis* is often very difficult on account of the vagueness of the symptoms, the absence of physical signs, and the resemblance of the pigmentation to that seen with various other cachectic states, especially cancer. *Cancer of the pylorus* is accompanied by sallowness, which is often mistaken for the pigmentation of Addison's disease. Both, moreover, are accompanied by enfeeblement, gastric pain, and vomiting. The diagnosis from *other pigmentary conditions* is given among the causes of pigmentation (§ 526). Slight jaundice, the pigmentation of malaria, chloasma, and arsenical pigmentation must be borne in mind. *Chronic Bright's disease*, neurasthenia, and other conditions attended by asthenia mentioned in this group are apt to be mistaken for the disease.

Prognosis.—The course of the disease is progressive, and usually prolonged ; it

[1] " On the Constitutional and Local Effects of Disease of the Suprarenal Capsules," London, 1855.

may last one to ten years. There are frequent relapses, with intermissions of comparative health, but it always ends in death. It may end suddenly with syncope, severe vomiting, and diarrhœa, convulsions, or coma, or it may terminate gradually by asthenia. The commonest complication is tuberculosis of the lungs, or elsewhere ; and pulmonary tuberculosis is the commonest cause of death. Occasionally cases run an acute course, death occurring in a few weeks.

Etiology.—Patients are usually about middle life, and by far the larger number are males. The essential cause is disease, often tuberculous, of the suprarenal capsules.

Treatment.—Suprarenal extract may be tried in tablets containing 1 grain (0·06) (equivalent to 15 (1 gm.) of the gland) twice daily, and in gradually increasing doses ; or adrenalin may be given subcutaneously. The pathology of the disease rests apparently upon the loss of the internal secretion of the suprarenal bodies, and if the secretion can be made good the fatal issue should be averted ; suprarenal extract and fresh gland, however, have not been successful in arresting the disease hitherto. The symptomatic treatment consists in rest, and supporting the strength by cod-liver oil, tonics, good nourishment and hygienic living, on the same principles as other tuberculous affections. Cold and over-exertion should be avoided.

§ 451. III. **Hæmochromatosis** (Bronzed Diabetes) is a rare disease, almost confined to the male sex, which seems to depend on some disorder of normal metabolism of iron, whereby the iron-containing pigment is not excreted but retained in the tissues. The liver, pancreas and skin suffer most, and the three cardinal symptoms of the disease depend on this fact. They are : (1) cirrhosis of the liver, with ascites ; (2) glycosuria, usually permanent and leading to coma, but in rare cases a subsidiary feature ; (3) pigmentation of the skin, giving rise to a slaty colour, occurring chiefly on exposed areas and not on the oral mucous membrane. The course is from a few months to one or two years, and treatment, except for the alleviation of diabetic symptoms or ascites, is unavailing.

IV. **Disease of the Pancreas, acromegaly, Graves' disease, myelopathic albumosuria, beri-beri, pellagra,** and other conditions mentioned in Groups I and II (*q.v.*), may come on with debility only, or the patient may seek relief for debility.

CHAPTER XVII

THE EXTREMITIES

IN the preceding pages we have seen on several occasions that so-called local diseases, such as pneumonia and endocarditis, have by scientific research been shown to be only local manifestations of a general microbic infection. This principle will here again be illustrated, for a gouty joint is only the local evidence of disordered metabolism, and acute rheumatism is probably microbic in origin. My own belief is that all joint diseases (other than traumatic) are but local manifestations of some toxic, septic, or infective blood condition. In conformity, however, with the scheme of this work, whereby all diseases are approached from a symptomatic standpoint, certain diseases, the symptoms and physical signs of which are referable mainly or entirely to the upper or lower extremities, will now be considered.

PART A. SYMPTOMATOLOGY

The CARDINAL SYMPTOM referable to the extremities is **pain** (or painful sensations of some kind), which may or may not be accompanied by some **physical change.**

§ 452. **Pain in the Limbs** should be investigated, like **pain** in other situations, as to its *position, character, degree, constancy,* and *duration.* Its position may be localised to the skin, or to a joint or any other structure, or be generalised, as in sheer exhaustion ; its character may be sharp and shooting (as in tabes) or dull and heavy (as in vascular lesions), or like pins and needles (as in nerve and neuro-vascular lesions). The skin, subcutaneous tissues, nerves, muscles, and vessels must be examined for a local cause ; but it must be remembered that pains in the limbs, especially in the legs, may be due to a generalised infection which may not be evident for some time after the onset of the pain. So also disease of the brain, spinal cord, chest, or abdomen may be the causal condition ; hence a thorough examination including investigation of the urine, blood and even lumbar puncture, may be necessary in obscure cases. Pain in the limbs may come on *acutely* or *insidiously.*

(a) *Acute pain in the limbs* coming on more or less SUDDENLY may herald influenza, enteric fever, malignant endocarditis, variola, scarlatina, or some other specific fever. In many cases of **influenza** this pain and

slight pyrexia are the only symptoms. *Acute rheumatism* also comes on rapidly with pains referable to the muscles, bones or joints, and so does *dengue* (" break-bone " fever). In *Trench fever* there is great pain and tenderness in the legs, especially in the shins. *Trichinosis* is attended by excruciating muscular pain in the second stage of the disease, when the parasite begins to migrate. Scurvy, osteomyelitis and epiphysitis are other causes of pain in the limbs associated with pyrexia. A sudden sharp pain in one spot in the limb is felt when *embolism* of an artery occurs ; so also in thrombosis of a vein. In either case pyrexia may be absent.

(*b*) Pains in the limbs coming on more or less *insidiously* may be due to (1) affections of the *nerves*, of central or local origin. Sciatica and peripheral neuritis are the most common causes. Long before such a disease as neuritis was recognised by the profession " pains in the limbs " were known to arise from excessive indulgence in alcohol, and in dealing with alcoholic subjects this should be remembered. The same pains may occur in neurasthenia, and it is quite possible that the pathological condition in this disease, as in alcoholism, neuritis, and the acute specific fevers, may be a toxic condition of the blood. Other causes of *nerve* origin are tabes dorsalis, cervical rib, cerebral tumour and meningitis, disease of the spinal cord or vertebrae, neuralgia, and acroparæsthesia. Severe pain in the foot should lead us to suspect flat foot or metatarsalgia. Metatarsalgia is a neuralgia of the foot due to lateral displacement of the heads of the metatarsal bones which press upon the nerves, and may also produce a corn (for which, indeed, the patient may seek advice). (2) Pains in the *joints* or *muscles* are characteristic of chronic rheumatism, rheumatoid arthritis, osteoarthritis, gout and all forms of *synovitis*. (3) *Vascular* affections giving rise to pain are thrombosis, embolism, intermittent claudication, erythromelalgia, aneurysm, varicose veins, phlegmasia alba dolens, Raynaud's disease, angina cruris and gangrene. The " numbness " and tingling is very characteristic, and may indicate only a vaso-motor disorder or it may be the incipient stage of tabes, general paralysis or other organic disease. (4) Certain *skin* diseases are accompanied by pains in the limbs. (5) *Growing pains* (so-called) in children are often of somewhat serious import, as being the only tangible evidence in them of subacute rheumatism, which may nevertheless be sufficient to produce endocarditis with permanent damage unless the condition is recognised and rest in bed with salicylates prescribed. (6) Various diseases of the *bones* (§ 477) may come on insidiously, with nothing more definite than vague pain in the limb or limbs. This must be especially remembered in children. Acute or chronic inflammation may arise, and unless the bone be superficial there may be no surface indications. Osteomyelitis is very serious, and requires prompt recognition. Syphilis causes pain in the bones, worse at night. Paget's disease, multiple myeloma, tumours, rickets, and blood diseases may cause pain in the bone. (7) A *muscular* strain or rupture of some muscular fibres may leave a chronic pain and partial loss of function (unattended by any physical sign) which is often hard

to cure ; in one of my cases the pain lasted over five years. Other causes of muscle pain are acute myositis, rheumatic fibrositis, trichinosis, tumours, and myositis ossificans. (8) *Local injury* or pressure may cause •pain, such as injury from a crutch, or sleeping in a cramped position, or lymphatic glands or other tumours in the axilla, neck, or pelvis. Heavy rings may cause ulnar neuralgia. Shooting pains down the arms, especially the left, occur in aneurysm of the aorta and angina (see also Causes of Single Nerve Paralysis, § 605). A careful examination of the chest should be made, for pain down the arm may indicate disease in that region ; *e.g.*, cardiac disease, aneurysm or other mediastinal tumour. Disease of the pelvis, vertebræ° and hip joint are frequently overlooked causes of pain in the legs.   •

PART B. PHYSICAL EXAMINATION

The physical signs referable to the extremities consist mainly of some visible or tangible alteration in the skin and general contour of the limb, the joint, the muscles, the bones, or the vessels and nerves.

§ 452. Inspection of the Limb may reveal generalised redness or alteration of colour, œdema, varicose veins, or some other diffuse or localised swelling. Eruptions prone to affect the extremities are dealt with in Chapter XVIII. Œdema of both legs is dealt with under Dropsy.

Even without the skill of a palmist or the acumen of a Sherlock Holmes a great deal concerning the temperament, habits, and diseases of a patient may be learned by a careful **inspection of the hands.** For instance, the long, thin, dextrous fingers, perpetually on the move, will almost surely indicate a nervous temperament and imaginative disposition, just as the short, thick, almost clumsy fingers and hands of another will bespeak slowness, deliberation, and doggedness. The occupation of a patient may often be learned from a glance at the palms. Some people habitually have cold, damp, clammy hands, and these are generally the subjects either of alcoholic habits or the rheumatic diathesis, occasionally some other condition causing a defective vaso-motor tone. The *nails* can also afford us some information. They are dusky in all conditions of impaired circulation, and pale in anæmia ; compression on the tip of the nail should not completely empty the capillaries, as it does in anæmia. In aortic regurgitation compression of the nail tip reveals a capillary pulsation. A transverse ridge or groove in the nails indicates an arrest of growth, and may mark the date of an illness or any disturbance of nutrition, of even so slight a nature as sea-sickness.[1] It is useful to remember that the nail takes about five or six months to grow from root to tip. Various distortions of the nail occur in neuritis and injury. Pitted, dark, and discoloured nails may be due to eczema, psoriasis, or ringworm. In the latter case scrapings of nail softened in liq. potassæ reveal the fungus. Clubbed fingers—*i.e.*, fingers with a bulbous end and great convexity of the nails (filbertshaped nails), are characteristic of congenital cardiac disease or valvular disease in early life. Pulmonary osteo-arthropathy, emphysema, chronic phthisis and any disease attended with profuse expectoration may be attended by the same deformity. *Glossy fingers* (fingers with smooth, thin skin) are the result of a neurotic dystrophy, and are associated with destructive and paralytic lesions of the nerve trunks ; they also occur in sclerodermia. Dactylitis is a thickening of one phalanx due to disease of the bone, with infiltration of the tissues of the fingers, resulting in a deformity known as the " champagne bottle finger." It is met with chiefly in tuberculous,

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[1] *Vide Illustrated Medical News,* about 1890.

and sometimes syphilitic, children. " Heberden's nodes," lipping and distortion of the phalangeal joints, are in reality osteo-arthritis of the fingers. Gouty nodules of urate of soda form white masses near the joints, just beneath the skin, and have an external resemblance to Heberden's nodes. The bone ends of the wrists are enlarged in rickets, syphilis, and pulmonary osteo-arthropathy. " Spade-shaped " hands (with thickened tissues) are suggestive of myxœdema, and large, flat, ungainly hands with osseous enlargement, of acromegaly and pulmonary osteo-arthropathy. The " claw-hand " (main en griffe) occurs as the result of injury or neuritis of the ulnar and median nerves; it is also seen in progressive muscular atrophy, syringomyelia, and cervical pachymeningitis. Wrist-drop is very characteristic of lead palsy.

CYANOSIS (BLUENESS) OF THE EXTREMITIES.—Blueness or redness of the hands and feet appear to be the evidences of a vaso-motor instability, want of vascular tone as a defective calcium metabolism. Such subjects are liable to chilblains. Often small doses of thyroid and calcium lactate aid these cases after any intestinal stasis has been corrected. A blue, rough and swollen extensor aspect of the arm is often a sign of pituitary deficiency. Slight degrees of cyanosis are revealed by examining the nails and depressing the nail tip. The *causes of cyanosis* were discussed in § 28. Erythromelalgia, " dead hands," gangrene, and Raynaud's disease are referred to below. Cervical ribs have been the causal factor in some cases.

§ 454. Varicose Veins consist of dilatation and tortuosity of the superficial veins, and are practically only met with in the legs, where their tortuous elevations produce obvious and characteristic alterations in the contour of the limbs. They occur chiefly in those who stand a

FIG. 116.—ELEPHANTIASIS TELANGIECTODES, in a woman about forty-eight years of age.

great deal, and are more frequent in the female sex, and especially in those who have borne children. Varicosity of the veins predisposes to eczema and ulceration, and severe hæmorrhage may ensue from their rupture. The *Treatment* of varicose veins belongs mainly to the surgeon.

Elephantiasis Telangiectodes (Fig. 116) is a rare condition which is apt to be mistaken for varicose veins. It consists of a hyperplasia of the subcutaneous tissues together with a varicosity of the superficial veins which form loose masses like bunches of grapes beneath the skin. They are usually associated with a certain amount of superficial telangiectasis (dilatation of venules in the skin) over and around the masses.

Gas gangrene, due to wound infection by B. perfringens, B. Hibler IX., and other dangerous organisms, begins as a localised swelling with crepitation and dark discoloration of the part. Surgical intervention may avert extension of the gaseous

swelling over the body. Without prompt surgery the gas extends rapidly over the body ; there is foul discharge, toxæmia, and death.

§ 455. Œdema of one Limb (localised dropsy) produces generalised swelling, which pits on pressure. The swelling due to the rare condition elephantiasis lymphangiectodes (see below) is of a much more solid character. Apart from *inflammatory œdema* and an extensive *angio-neurotic œdema* (§ 485), dropsy of one arm or one leg always points to some obstruction of the main vein of the limb by thrombosis within or pressure upon the vein. (1) *Thrombosis* (coagulation within the living vessel) with or without phlebitis (inflammation of the vein) is not uncommon in the femoral or iliac vein in the leg, and the brachial in the arm. In addition to œdema there are pain and tenderness at the seat of the obstruction, and a history or evidence at the time of some cause of ethrombosis or phlebitis, such as phthisis and other wasting disorders, any of the acute specific fevers, injury or local extension (as from an ulcer). The commonest example of thrombosis is *phlegmasia dolens* (or white leg), which is so apt to come on after confinement, partly as a result of the hyperinotic condition of the blood (i.e., the excess of fibrin-forming constituents) which is associated with the puerperal state, and partly owing to previous pressure on the veins within the pelvis. Little can be done in the way of treatment beyond complete rest in the horizontal position, warmth applied to the limb, and the administration of iron. Nothing will remove the obstruction, but in course of time the condition is relieved considerably, if not altogether, by the establishment of collateral circulation. (2) Œdema may also be due to *pressure upon a vein* by a tumour, such as enlarged glands in the axilla or elsewhere, aneurysm, or other intra-thoracic growth pressing upon the veins coming from the arm ; pelvic cellulitis, carcinoma of the uterus or bladder, bands of adhesion, hydatid, or other intrapelvic growth pressing on the veins of the leg. Local tenderness is present in association with œdema in thrombosis, scurvy, and trichinosis.

FIG. 117.—ELEPHANTIASIS LYMPHANGIEC-TODES in a man about forty years of age who had never been abroad.

Elephantiasis Lymphangiectodes (Fig. 117) is a solid œdema, not pitting on pressure in any notable degree, affecting one leg, occasionally one arm, or the scrotum, due to a blocking of the lymphatics of the limb. It is met with chiefly in tropical countries in persons whose blood contains the filaria embryos. The adult worm is believed to block the lymphatics, and so produce the disease. It is, however, occasionally met with in temperate climates in persons whose blood does not reveal the parasite, and the cause of the blocking in these cases is obscure. In some cases this œdema has affected several members of a family (Milroy's disease). It may be unilateral, but more often affects both lower extremities. There is no fatal tendency. The cause is unknown.

§ 456. Swelling of the Lymphatic Glands in the neck, axillæ, groins, or elsewhere on the surface of the body or limbs may be due to : 1, injury and septic or infective processes ; 2, tuberculous disease ; 3, malignant disease ; 4, syphilis ; 5, acute specific

fevers; 6, leukæmia; 7, Hodgkin's disease; 8, glandular fever; 9, plague; 10, trypanosomiasis; 11, Japanese river fever. The first three arise in glands adjacent to some focus of mischief, and the glandular swelling usually remains localised; in the remainder all the lymphatic glands tend to become affected.

1. *Local* injuries, septic sores, infected tonsillar crypts and abscesses give rise to enlargement of the neighbouring lymphatic glands. Pain and enlargement of the glands in the groin, for instance, may be due to direct injury to or pressure on those glands ; but one should always carefully inspect the foot for abraded skin around the toe-nails, through which dirt or stocking dye may have been aborbed. *Post-mortem scratches* or inoculation from septicæmia cases are of a much more virulent nature. Red streaks along the course of the lymphatics indicate lymphangitis. The glands at the elbow and axilla become acutely painful and tender, and they may rapidly suppurate. This is a conservative process, for in this way the septic virus is prevented (usually) from extending to the general circulation. If, however, the virus is too intense or the dose be too large, general septicæmia and death in a day or two is the result before suppuration can occur.

2. *Tuberculous* disease of the lymphatic glands, especially of the neck, is very frequent in children. The disease is usually secondary to some other focus latent or active, but is generally localised to one group of glands, and the process is slow and chronic. It is also recognised by the fact that the glands very soon become matted together into one solid mass, which in due time, if the case be untreated, undergoes caseation, breaks down, and leaves a characteristic ulcer.

3. *Cancer* gives rise first to inflammatory enlargement of the adjacent glands ; later, the adjacent and distant glands become the seat of secondary cancer (§ 445). Lymphosarcoma is a sarcomatous growth *starting in* the lymphatic glands. It rapidly invades the surrounding structures, and the neighbouring glands.

4. *Syphilis* first affects the lymphatic glands in the neighbourhood of the chancre. They are small, hard (shotty), painless, and only perceptible on palpation, but for many years afterwards all the glands of the body, especially those in the groin, may be discovered on careful palpation to be thus indurated. They never suppurate with syphilis as they do with a soft chancre.

5. In most of the *acute specific fevers* there is, as in syphilis, a slight generalised glandular enlargement. In those fevers which have a local manifestation—the throat in scarlatina and diphtheria, for instance—the adjacent glands are first and chiefly affected. In German measles the occipital glands are especially noticeable. In bubonic plague the enlargement is very large. In certain milder cases of *plague* nothing but slight glandular swelling and a little fever occurs (pestis minor). *Rheumatoid arthritis (Still's disease)* is accompanied by enlargement of the glands and spleen, especially in children.

6. In *Leukæmia* there is a generalised enlargement, and the blood changes are characteristic (§ 437).

7. *Hodgkin's Disease* starts with a swelling of one group of glands, which enlarges paroxysmally, generally attended with corresponding paroxysms of fever. The individual glands remain separate, painless, and may feel like a bunch of grapes. Each gland may attain a large size. Sooner or later other groups of glands become similarly involved (§ 438).

§ 457. The JOINTS, MUSCLES, BONES, VESSELS, NERVES, and CONSTITU-TIONAL SYMPTOMS should be next investigated.

The joints may need investigation for tenderness, pain, heat, swelling, or redness, and for loss of function or range of movement. The affected and the unaffected sides should be carefully compared. Slight degrees of fluid in a joint are often difficult to detect. The active movements (those which the patient can make) and the passive movements (those made by the doctor) should, with due consideration and caution, be tested. Among the *fallacies*, paralysis, or muscular weakness is often simulated

by chronic joint diseases, and *vice versa*, and pain in the limbs from various causes will often simulate a stiffness of the joint. Disease near a joint may be mistaken for a diseased joint. Pain may be referred, *e.g.*, in hip-joint disease pain is often complained of at the knee. In neuritis pain may be referred to the joint supplied by the affected nerve. X-rays may aid. In acute joint disease the fallacies of epiphysitis and acute osteomyelitis must be avoided. The presence of associated symptoms may aid a diagnosis ; for example, tophi suggest gout ; purpura and subcutaneous nodules, rheumatism.

The **muscles** may be investigated for tenderness, stiffness, or swelling. The investigation of paralysis, tonic or clonic spasm, or wasting, is given under diseases of the nervous system (Chapter XIX). We are here concerned only with pain, tenderness, or swelling localised in the muscles ; it is the presence of these localised symptoms which helps us to differentiate muscular diseases from paralysis and other diseases of the nervous system. To decide that the lesion is not in the bones or ligaments may be difficult ; if it be in the muscle, the pain is greater during active than passive movement of the affected muscle ; if in the ligaments or joints, the pain is about equal.

The examination of the **bones** belongs specially to the surgeon, but disease situated in the bones may be evidenced by pain, tenderness, swelling, or deformity. They often first come under the notice of the physician when pain is their only symptom, and the diagnosis presents considerable difficulty. X-ray examination is indispensable.

In the diagnosis of SWELLINGS CONNECTED WITH BONES it is well to remember the following data. Symptoms come on *acutely* with trauma, periostitis, osteomyelitis, and deep abscess ; slowly and *chronically* with caries, necrosis, chronic periostitis and osteitis, rickets, syphilis and tumour. In regard to physical signs the *diaphysis* is mainly affected in acute and chronic inflammation, in sarcomatous and other tumours ; the *epiphysis* in rickets, syphilis, and central sarcoma. The consistency of the swelling is *soft* in abscess and vascular sarcomata, *hard* in chronic inflammation. As regards the mode and rate of growth, the swelling *progressively enlarges* in inflammatory and malignant tumours, and is *stationary* in chronic inflammation and benign tumours ; *receding* swellings are always inflammatory.

The **vessels** and **nerves** need examination when any of the symptoms indicate their implication, as in erythromelalgia and some other conditions in Group I below. Pressure along their course may elicit tenderness, indicative of inflammation. The symptoms and effects of peripheral neuritis are given in Chapter XIX, and embolism of an artery or thrombosis of a vein in § 460.

The **viscera** should be examined, particularly in acute joint diseases, which are almost always the product of some blood disorder—*e.g.*, the heart must always be examined in rheumatic conditions, the kidney in gouty disorders.

Pyrexia and **Constitutional Symptoms** are present in a considerable number of diseases of the extremities, particularly in the acute joint and bone disorders, and they may be investigated on the lines laid down in Chapter XV. Rigors and sweating indicate a pyogenic process. Characteristic blood changes are found in several diseases, notably glandular and septic processes.

PART C. DIAGNOSIS, PROGNOSIS, AND TREATMENT OF DISEASES CAUSING SYMPTOMS REFERABLE TO THE EXTREMITIES

§ 458. Routine Examination and Classification.—As a matter of routine, as in other cases, investigate—

First, the LEADING SYMPTOM, which in this instance is very often as visible or palpable to the patient as to the physician.

Secondly, the HISTORY of the case, its mode of onset (acute or chronic), and evolution in chronological order.

Thirdly, examine the AFFECTED LIMB or limbs, their colour and contour, the joints, muscles, bones, vessels, or nerves, as may be indicated; and, finally, examine the VISCERA and the TEMPERATURE. The sensation, movements, and reflexes should be tested in cases where nervous disease is suspected. An X-ray examination is useful in obscure cases.

If there is any visible abnormality in the COLOUR of the hands or limbs, turn to Group I, below.

If the symptoms point to JOINT disease, acute or chronic, turn to Group II, p. 653 (Acute), or p. 663 (Chronic).

If the symptoms point to disease of the MUSCLES (rare), turn to p. 673.

If the symptoms point to disease of the BONES, turn to p. 676.

If the symptoms point to disease of the NERVOUS SYSTEM, turn to § 588.

GROUP I. ALTERATIONS IN COLOUR OF THE EXTREMITIES

This group comprises only the following morbid conditions which may be considered medical. Other alterations in colour or contour, such as œdema of one limb and varicose veins, have already been referred to in §§ 453 and 454. Pigment alterations are described in § 526. There remain—

1. Erythromelalgia and Acroteric Scleroderma.
2. Gangrene. -
3. Raynaud's disease.
4. Dead hands.
5. Intermittent claudication.
6. Cyanosis, clubbed fingers, etc. (§ 453).

§ 459. **Erythromelalgia** (a term first used by Weir Mitchell) is a painful redness and swelling occurring in paroxysms, and symmetrically affecting both hands, sometimes the feet, and sometimes spreading to the arms and legs. One side may be more affected than the other, but I have not seen any cases in which both sides have not been involved to some extent.[1] The disorder starts intermittently with tingling and numbness in the extremities (acroparæsthesia), and later on a painful redness supervenes. The paroxysms are often determined, and always aggravated by hanging the limbs down, and also by placing them in very hot or very cold water. They are often worse when the patient lies down and goes to sleep, and thus the night may be badly disturbed. The pain and swelling are lessened by holding the hands over the head, or raising the feet. There is no paralysis, but owing to the numbness and swelling the fingers cannot easily be bent. The swelling and redness affect the whole hand (Figs. 118 and 119)—not patches, as in chilblains, lupus, or erythema. On this account the *Diagnosis* is not difficult. In the cyanotic form of *Raynaud's disease* the symptoms start and prevail in one or two finger tips; in erythromelalgia all the fingers and the whole hand are about equally involved. Thickening of the subcutaneous tissues may ensue, and the paroxysms are apt to return even after long intervals. It is a prolonged and very painful disorder, but it is not fatal, and is in my experience to some extent amenable to treatment.

Etiology.—The female sex is far more prone to the disease; out of thirty-seven consecutive cases which I observed between 1900 and 1902 only two were males. It appears to arise more frequently between the ages of eighteen and twenty-five, and at the climacteric. The rheumatic, gouty, and hysterical diatheses predispose to the complaint, and the determining cause is, in my belief, some altered condition

[1] See a clinical lecture on this subject in the *Lancet*, June 1, 1902; and "Lectures on Hysteria," Glaisher, London, 1906.

of the blood. It certainly occurs with erythræmia and some cases of chlorosis. Several cases which I have observed have exhibited, concurrently with a severe paroxysm of the erythromelalgic symptoms, erythematous blotches on other parts of the body, and severe " rheumatic " pains in the limbs. The disease must, I think, be regarded as a vaso-motor paralysis due to a toxæmia, possibly focal sepsis in some cases.

Treatment.—Bromides invariably relieve the condition for a time ; arsenic, strychnine, quinine, and other tonics are useful. Ergot sometimes does good. The general health should be attended to, and particularly the digestion. Septic foci must be removed. I have tried salicylates once with success. A weak descending galvanic current is the most effective curative agent in my experience.

Acroteric Scleroderma (Hutchinson) or Sclerodactylia is a scleroderma affecting the hands and feet, and sometimes the nose, in which the skin is bluish and thickened at first, white and atrophic afterwards.

§ 460. Of Gangrene, necrosis, or death of part of an extremity, there are two kinds :

(a) In DRY GANGRENE the extremity becomes white and cold, then of an ashy

FIG. 118.—ERYTHROMELALGIA in a woman aged about thirty.

FIG. 119.—ERYTHROMELALGIA, showing maximum closure of the hand.

and black colour ; the part shrivels up, becomes dry and mummified. It is chiefly met with among old people—senile gangrene—and is due to the gradual obliteration of the lumen of the artery supplying the part, combined with more or less cardiac enfeeblement. It is met with in younger patients in Raynaud's disease, claudication and thrombo-angiitis obliterans and in cases of embolic blocking of an artery. The artery is tender at the seat of the embolism, and ceases to pulsate below.

(b) In MOIST GANGRENE the part becomes cold, purple, or mottled, and engorged with blood. Blebs then form on the surface, and a bright red line separates the dead from the living tissues. The dead part ultimately sloughs off, and leaves an ulcer. This gangrene is due to venous obstruction, the result of thrombosis, pressure, injury, or inflammation. The gangrene occurring in diabetes is of the moist variety. The treatment of both of these conditions belongs to surgery, but warmth, the posture of the limb, and the administration of diffusable stimulants are points to be attended to.

Trench Foot is an affection of which the etiology is uncertain, but in which cold, wet, and more especially blood stasis appear to be causative factors.

Symptoms.—The onset is usually slow, following exposure to conditions named.

Then development of (1) great pain and hyperæsthesia with practically no objective symptoms, confined as a rule to the feet but sometimes affecting the hands also ; or (2) œdema of the feet, with, as a rule, much less pain; or (3) either of above may eventuate in gangrene.

Treatment.—Rest, massage, application of heat, galvanism, and static effleuve have all a share in promoting recovery, but patients differ widely in their reaction to therapeutic measures. Recently subcutaneous injection of oxygen into the foot below the ankle has given good results. Opiates may be required for pain.

§ 461. **Raynaud's Disease** (Synonyms : Symmetrical Gangrene, Local Asphyxia of the Extremities).—This disease, which was first described in 1862 by Dr. Raynaud,[1] is characterised by local vascular changes in one or môre of the fingers, for the most part symmetrically on the two sides of the body, resulting very often in gangrene. Three types or stages of the disease have been described—a syncopal type, due to vascular spasm, an asphyxial type, due to vascular dilatation, and a gangrenous type.

Symptoms.—First is noticed pallor (*local syncope*) and numbness of one or more of the fingers or toes, usually the corresponding finger or toe on both sides, coming on in attacks, lasting an hour or more. This pale stage is generally followed by a reactionary stage of congestion and heat with swelling and lividity (local asphyxia), in which the tip of one or more of the fingers or toes, or the ears, may be dark purple. There is usually a good deal of pain. Sometimes the pale stage is very definite, sometimes it is wanting, or it may be so transient as to be unobserved. Occasionally the entire hands are involved. After a number of these attacks *gangrene* occurs at the area affected, the dead becomes separated from the living part, and the ulcer that is left heals normally, but slowly. Cases have been recorded of extensive multiple gangrene in which the patient has lost entire limbs in this way. The attacks described may be the only symptom, but in most cases other symptoms of considerable pathological interest may be observed. In some there is a generalised scleroderma, the skin having the appearance of being stretched, and smooth, or sometimes cracked ; in such cases all the fingers are pale and dead-looking, and their entire substance becomes wasted. In other cases erythematous blotches occur from time to time in different parts of the body, leaving bruise-like stains. The patients are usually nervous, and prone to emotional attacks. Transient attacks of hemiplegia and aphasia have been observed, also of paroxysmal hæmoglobinuria, all pointing to vaso-motor irregularities. Effusion into the phalangeal and other joints may supervene, and may result in ankylosis.

The *Diagnosis* is not usually difficult. In the earlier stages it is closely allied to erythromelalgia, sclerodactylia, and to " dead hands," but these affections are not so localised to the fingers' ends, are less severe, and never go on to gangrene. Local vaso-motor symptoms, affecting usually only one arm, may be due to a *cervical rib* (§ 606).

Prognosis.—The disease runs a prolonged course of many years. The attacks become more prolonged and frequent, and the patient gradually becomes more and more helpless. There are many degrees of severity of this disease, ranging from what amounts to no more than a small localised syncope or asphyxia to gangrene of the entire segment of a limb. It is a curious circumstance which I believe to be uniform, that once a finger has become gangrenous the stump does not become similarly affected later on. The subjects of this malady in a marked form rarely reach old age, but usually die of some intercurrent malady.

Etiology.—The disease is more common in women, and especially those of a nervous diathesis. It appears for the first time usually between the ages of fifteen and thirty. Attacks may be brought on by chill or mental disturbances. The pathology of this strange disorder is unknown, but it is undoubtedly a vaso-motor affection, and in view of its symmetry is very probably a derangement of the local vaso-motor centres in the cord. Endarteritis in the peripheral vessels has been described in a few cases.

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[1] " Thèse de Paris," a thesis written for the M.D. at the Paris University in 1862.

Treatment.—The affected limbs must be kept warm with cotton-wool, and the patient protected from exposure to cold. The most efficacious remedy is undoubtedly electricity ; a strong descending galvanic current does good. Thyroid gland is beneficial and nitroglycerine has been used in the syncopal type. The pain is intense, and may require morphia, which acts in a double way in asphyxial cases by giving tone to the vessels. Einhorn claims success with duodenal lavage.

§ 462. **Dead Hands** (Pallor of the Fingers).—Many patients—but particularly those who present other evidences of an inherent vaso-motor instability, and are subjects of the gouty or rheumatic diathesis—complain that the hands or finger-tips "go dead," or white, like those of a corpse, and feel numb and cold. These attacks, which rarely last very long, may happen in warm summer weather, without any obvious cause, or consequent on anything which produces a nervous or emotional condition. This vascular disorder appears clinically to be the converse of erythromelalgia, the fingers or hands being pale or shrunken instead of red and swollen. It is akin to acroparæsthesia and erythromelalgia, but it most resembles the slight or early phase of Raynaud's disease. These attacks are not as a rule serious. As they often depend upon oral or gastro-intestinal sepsis treatment is directed to the underlying cause, and massage and electricity are given locally.

§ 463. **Intermittent Claudication** (Intermittent Limping) is a rare condition occurring chiefly in men. It is due to spasm or sclerosis of the arteries supplying the affected leg, usually of the smaller branches, but sometimes of the main trunks. The symptoms come on when an extra local supply of blood is required, such as during walking exertion. The symptoms are cramp-like pains associated with pallor or blueness, and the leg goes cold, numb, and powerless. During an attack the pulse of a distal vessel (*e.g.*, the dorsalis pedis) may cease to beat. Sometimes gangrene supervenes. The cause is unknown ; syphilis and tobacco have been blamed. *Thrombo-angiitis obliterans* affects both veins and arteries, and is probably an allied toxic condition.[1] There are four indications for *treatment* : (i.) To treat the arterial sclerosis. This is only effectual in the case of syphilis.[2] (ii.) To avoid undue exercise or fixation of the affected limb ; (iii.) to avoid cold ; (iv.) to increase the peripheral blood-supply by vaso-dilators such as nitroglycerine, by warmth, and by massage or galvanism. Nitrites are useful, especially for acute pain.

GROUP II. JOINT DISEASES

The methods of examination and exclusion of fallacies have already been described. Arthritic disorders may conveniently be grouped into acute and chronic :

Acute.	*Chronic.*
I. Acute gout.	I. Chronic gout.
II. Acute rheumatism.	II. Chronic rheumatism.
III. Acute gonorrhœal arthritis.	III. Rheumatoid arthritis.
IV. Acute rheumatoid arthritis.	IV. Osteo-arthritis.
V. Pyæmia.	V. Spondylitis deformans.
VI. Acute specific fevers.	VI. Gonorrhœal arthritis.
VII. Purpura, scurvy, hæmophilia.	VII. Chronic septic processes.
VIII. Traumatism.	VIII. Tuberculous synovitis.
IX. Extension from adjacent bone.	IX. Syphilitic arthritis.
	X. Hysterical joint disorder.
	XI. Neuropathic joint disease.

[1] Four cases are narrated in the *Brit. Med. Journ.*, Dec. 6, 1924.
[2] One case under my care proved on investigation to be a typhoid carrier, and was cured by an autogenous vaccine (Agnes Savill).

(a) Acute Joint Diseases

Acute joint diseases, a list of which has just been given, come on more or less abruptly, and are as a rule attended by the local and general signs of inflammation. Acute rheumatism is essentially an erratic polyarthritis from the commencement; acute gout usually affects a single joint; most of the other causes start in one joint, but (excepting VIII and IX) tend to a progressive involvement of others. It is worth noting that all the acute joint disorders (traumatism being excluded) are due either to some microbic process or to some other blood disorder. These facts emphasise the necessity of investigating the constitutional symptoms, the viscera, and the blood.

§ 464. I. **Acute Gout.**—Gout is a diseased or disordered metabolism associated with excess of uric acid in the blood, and characterised by recurrent attacks of acute inflammation of the joints with deposition of sodium urate. It is one of the oldest known diseases, but is at the present time quite rare. Gout occurs in acute, chronic, and irregular forms.

The *Symptoms* of an attack, or paroxysm, of acute gout are usually preceded by gouty dyspepsia, heartburn, flatulence, and weariness after food, fulness and tenderness in the epigastrium and liver, indentation of the tongue, a bad taste in the mouth, and excessive secretion from the fauces in the morning, scanty, high-coloured urine constantly depositing urates, cardiac irregularities and intermission in the pulse, restlessness at night, and a tendency to catarrh of the mucous membranes on the slightest exposure. The onset of an attack is usually *very sudden*, often in the middle of the night. It affects preferably one of the smaller joints, and especially the metatarso-phalangeal joint of the big toe. The swelling in a marked case is tense, shining, red, pits on pressure, and is acutely tender, but suppuration never occurs. Other joints may become affected, but the inflammation does not shift from one joint to another as in acute rheumatism. Mild constitutional symptoms are present with pyrexia (102°); there may be muttering delirium at night. The urine contains less uric and phosphoric acid before the attack, and more during it, and may contain a trace of albumen. An attack lasts from two to three days or two to three weeks. After an attack the health is frequently better than it was previously; but the intervals between the attacks gradually become shorter as time goes on, at first two or three years, then one year, then six months; finally the disease becomes chronic, and permanent changes take place in the joint. *Chronic gout* and its associated symptoms are described under chronic joint diseases, § 467.

Varieties.—(1) The symptoms of *irregular gout* consist chiefly of dyspepsia and a variety of symptoms referable to various organs of the body, supposed to be due to a deposit of gouty materials therein (see complications below). (2) *Retrocedent* or suppressed gout is a term applied to cases where the joint mischief suddenly improves coincident with internal symptoms affecting the digestive tract, the heart or the brain.

Thus there may be vomiting and diarrhœa, dyspnœa, arrhythmia, even pericarditis, delirium and coma, or cerebral hæmorrhage. These symptoms are often associated with chronic interstitial nephritis, which is one of the consequences of gout.

The *Diagnosis* of acute gout is not difficult except sometimes from acute rheumatism.

TABLE XXVI.—DIAGNOSIS BETWEEN ACUTE GOUT AND ACUTE RHEUMATISM.

Acute Gout.	Acute Rheumatism.
In typical cases : Middle age ; male sex.	In typical cases : Youth ; either sex.
Preference for smaller joints ; never wandering from joint to joint.	Preference for larger joints ; usually wandering from joint to joint.
Swelling is usually red, tense, pitting on pressure, acutely tender. Pain persists during rest.	Swelling is hot, but pale ; pain only on pressure or movement of joint.
Ears show tophi.	No tophi.
Fever may be slight or transient.	Fever always marked and continuous.

Prognosis.—The duration of an attack or paroxysm—which is rarely fatal in itself, depends mainly upon the age and constitutional condition of the patient. When, however, symptoms of suppressed gout come on, the case may end fatally with great suddenness. Gout tends to shorten life mainly by the resulting kidney disease and cardio-vascular changes, and the ultimate prognosis largely depends upon the condition of the urine, which should be of good specific gravity and free from albumen. Among the complications and so-called *irregular* forms of gout (1) chronic interstitial nephritis is the most important. During an attack there is generally a certain amount of albuminuria owing to congestion of the kidney, or deposit of urate of sodium, but this passes off. Gradually, however, after repeated attacks, an interstitial fibrosis takes place in the kidney, which from this association is known as the gouty kidney. Glycosuria occasionally occurs. Renal calculus may occur in persons of the gouty diathesis who may have escaped joint symptoms. (2) Cardiac and cardio-vascular diseases come next in frequency. The " gouty " heart is one which is " irritable," acts irregularly, causing palpitation and pain, and often great distress. Tachycardia is common. Various valvular lesions and a thickening and degeneration of the arterial walls occur. (3) Bronchitis of a subacute or chronic form is frequent, and in treating this the gouty condition must not be forgotten. (4) Various gastric and hepatic derangements are frequent in gouty subjects. Gastrodynia or an agonising pain in the abdomen is sometimes caused by indiscretions in diet. (5) Eczema and other skin diseases of an intractable kind super-

vene. Hot, itchy eyeballs, migraine, and episcleritis are often present. Glaucoma and iritis also occur.

Etiology.—Among the *predisposing* causes of gout, age, sex, and heredity are extremely important. (1) The disease is rarely met with under thirty, and the tendency increases up to the age of fifty. It rarely starts for the first time over that age. (2) The disease is almost confined to men; if it occurs in women, the attacks are generally very slight. (3) Out of 520 cases collected by Sir Alfred Garrod, 332, or nearly two-thirds, were distinctly hereditary. The predisposition, like landed possessions, is transmitted mainly through the male line; but rarely it may be transmitted by an unaffected female, and reappears in the sons. (4) Lead in the system is also a strong predisposing factor; painters, glaziers, etc., are very prone to gout and gouty kidney. (5) Attacks are more frequently met with in the changeable weather of the spring and autumn. The *exciting* causes are (1) the use of alcohol, and especially those forms which contain a high percentage of both sugar and alcohol, such as port wine, brown sherry, Madeira, sweet wines generally, and malt liquor. (2) Nitrogenous food in excess, especially the purin-containing organs such as the liver and kidney, is credited with being able to produce gout; at any rate, gout is commoner in butchers and meat eaters than among vegetarians (compare remarks below). (3) Both of the foregoing causes are more potent when combined with deficient exercise in the open. (4) It is a curious and imperfectly explained fact that gout is very rare among Scottish artisans. A possible explanation of this exemption is that the beverage of the Scottish artisan is whisky, while that of the English workman is beer, which contains over a grain per pint of purin bodies. An attack may be determined by (1) a debauch of alcohol, especially of certain kinds; (2) indigestion; (3) a chill to the surface of the body; (4) severe mental or bodily fatigue; (5) injury to a part, which will not only determine an attack but also the particular part affected. There is, however, a tendency for attacks to recur in the same joint.

, The discussion of the pathology would be out of place here, but it may be repeated that the clinical phenomena are due to the presence of uric acid in abnormal amount. Meat in itself is not a cause of gout, and in many gouty subjects does not cause gout except when taken with carbohydrates which cause fermentation in the digestive tract. Recent research tends to prove that in the gouty individual the liver cannot deal with the oxidation of purin bodies to urea as it does in health. Even on a purin-free diet the liver of the gouty individual cannot oxidise the endogenous purins. Dr. Langdon Brown expresses it thus: " The gouty subject seems unable to metabolise his purins properly, and his kidneys excrete them too slowly. The inadequate metabolism leads to toxic symptoms, and the inadequate excretion leads to uratic deposit."

Treatment during an attack.—(1) A low diet of milk and farinaceous food, and complete abstinence from alcohol should be enjoined, unless the heart be weak, when well diluted pure spirit is the only form permissible. (2) A brisk cathartic with one or more grains of calomel should be given at the onset, followed by frequent doses of saline purgatives, such as Hunyadi Janos water, or Carlsbad. (3) Alkaline carbonates

(potassium, lithium, sodium) promote the solution of uric acid. (4) Colchicum (combined with alkaline carbonate) is regarded as a specific, and it may be given every four hours (M 40 (2·3) for first dose, then M 12 (0·8), or F. 88) until the pain is gone; then it should be stopped. (5) Opium may be needed for the pain, but should not be given if there be albuminuria or other evidence of renal changes. (6) Local treatment consists of complete rest, wrapping the joint in cotton-wool, and the application of sedatives to the joint, such as lotions of sodium bicarbonate Ʒiv (16), with laudanum Ʒii. (8) in Ʒx. (320) of water. A very comforting lotion consists of sp. vin. rect. ; liq. amm. acet. ; aq. rosæ āā Ʒiii. (12) ; aq. ad. Ʒxii. (384). When symptoms of suppressed gout come on, employ eliminatory treatment promptly, stimulate if symptoms of collapse follow, and apply counter-irritation (mustard, turpentine stupes) and hot fomentations to the chest or abdomen as the case demands.

Treatment between the attacks—i.e., preventive treatment, resolves itself mainly into a question of diet, and the treatment of gouty dyspepsia. All foods rich in purin bodies are debarred, such as sweetbread, liver, roes, stock soups, and meat essences and extracts. The flesh of young animals is worse than that of old. Harmful vegetables are asparagus, peas, and beans. Purin bodies also occur in tea, coffee, cocoa, chocolate, and beer. The diet should be light and excess of sugar avoided. Sweet wines, malt liquors, pastries, and over sweet and greasy dishes should be forbidden. Plain food and abundance of pure water (to aid elimination) must be ordered. Light claret and dry sherry are allowed in moderation. Whey retards uric acid formation, possibly by its diuretic effect ; 2 or 3 cupfuls may be taken daily. A " uric-acid-free " diet consists of bread, macaroni, rice, and other cereals, biscuits, milk, cheese, nuts and almonds, dried fruits, and most vegetables. Fresh fruit does not suit many gouty persons. Water should be drunk freely and open-air exercise strongly enjoined. Among the remedies for the elimination of uric acid the most valuable is atophan, 15-grain (1 gm.) doses three times a day. Phenoquin is the English equivalent ; 4 to 6 tablets should be taken daily, with half-dram doses of bicarbonate of soda, in plenty of water. Solurol (thyminic acid), hexamine and piperazine are also recommended. An occasional dose of mercury followed by a saline is useful. Colchicum is of great use in the subacute exacerbations, when it may be given with large doses of potassium iodide until the pain is relieved. Mineral waters (Carlsbad, Vichy, Hunyadi Janos, Friedrichshall) can be freely taken. Some advocate the use of potassium instead of sodium salts, and cases are reported which remained free of gouty symptoms when taking potassium chloride instead of common salt with meals. Visits to Bath, Harrogate, Buxton, and Strathpeffer, in this country, are undoubtedly beneficial. Carlsbad, Royat, Aix-les-Bains, and a number of other foreign spas are annually visited. Gouty dyspepsia may be treated by the mineral waters just mentioned, or on the principles laid down in Chapter X. Locally, blisters or iodine may

C.M.   U.U

be employed near the joints ; or alkaline lotions (carbonate of lithium or sodium, gr. 10 (0·6) to the ounce (32)).   Massage and radiant heat treatment is also useful (see Chronic Rheumatism).

§ 465. II. Acute Rheumatism (Rheumatic Fever) is an acute febrile disease, with erratic painful swellings of the joints and a marked tendency to disease of the heart ; running a prolonged course of many weeks if untreated, and followed by a great tendency to relapse.   It is a disease especially of childhood, when it is capable of many manifestations.   The poison of rheumatic fever tends to affect not only the joints, but also all the fibrous, serous and muscular tissues.   The serous membranes of the joints, endocardium, and pericardium (which, it will be observed, histologically resemble each other) are the favourite situation of the inflammation.   Acute rheumatism, unlike acute gout, attacks several joints, usually
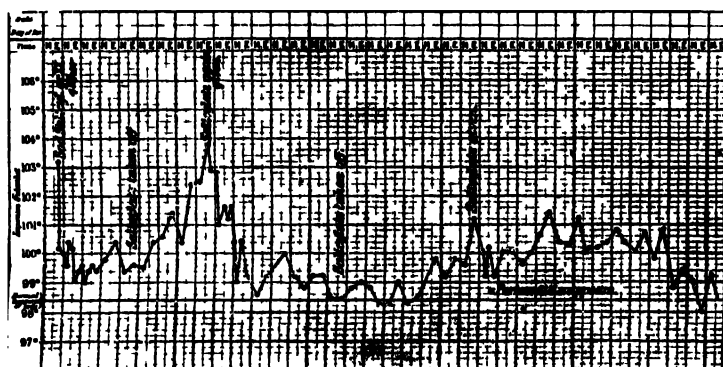
FIG. 120.—RHEUMATIC FEVER —Henry H——, æt. twenty-two · the chart shows efficacy of sali-cylates in reducing the temperature until pericarditis appears; then the controlling power of the drug is less.

the larger ones—e.g., the knees, ankles, shoulders.   In adults it occurs only in a modified form, with polysynovitis as its most distinctive feature.

Symptoms.—(1) The fever, which may have been preceded by tonsillitis for a day or two, comes on in the course of twenty-four hours, setting in before or at the same time as the joints are inflamed.   It is of a continued type (Fig. 120), usually remaining about 102° or 103° F. for some days.   The onset of any inflammatory complication in the pericardium or elsewhere is marked by fever, pain, and sometimes delirium, which is otherwise extremely rare in acute rheumatism ; in uncomplicated cases the mind remains quite clear throughout.   The usual accompaniments of pyrexia are present—viz., the urine is scanty, highly coloured, loaded with urates, with an excess of urea and deficiency of the chlorides, the tongue is coated, the pulse quick and bounding, usually over 100.   The blood exhibits considerable leucocytosis.   (2) In adults there is a profuse perspiration with a sour disagreeable odour and an acid reaction, but in children this is unusual ; later on sudaminal vesicles are frequently seen.

Erythematous, purpuric, and other *rashes* occasionally appear. (3) The two distinguishing features of the *joint lesions* of acute rheumatism are their wandering or metastatic character, and the absence of suppuration. The effusion into a joint is not very great; first one joint is affected, but the next day another is involved, the first joint having almost recovered; finally several may be affected together. The joints are hot and swollen, and though not tender to the touch are acutely painful on the slightest movement. The skin over the joints is either unaltered in colour or shows a faint flush. (4) *Peri-* or *endo-carditis* are other manifestations of the disease; the pericardium may be the first serous membrane to be affected. In 150 fatal cases analysed by Dr. F. J. Poynton, evidence of mitral endocarditis existed in 149. There is always some *dilatation of the heart* in rheumatic fever, which is due to the action of the toxin on the cardiac muscle. *Myocarditis* is commonly present, but rarely occurs as the sole cardiac lesion. (5) *Rheumatic nodules* occasionally occur. They are small movable bodies, usually fibrinous, but may become fibrous. They are generally symmetrically placed on opposite sides of the body and appear on bony prominences and prominent tendons. The commonest places to find them are about the elbows, knees, malleoli, occipital curved lines, posterior spinous processes of the vertebræ, and knuckles. (6) *Chorea* may be the first and only sign of rheumatism, but is frequently followed by arthritis and endocarditis. (7) Pneumonia, pleurisy, iritis, periostitis, peritonitis, and meningitis, all occur rarely. (8) In untreated cases the fever and local inflammation may subside gradually in four to five weeks, and return again after an interval lasting, perhaps, a few days to a fortnight. Even after recovery the liability to recurrence is very great, and special care is needed. In no other acute specific disease, excepting perhaps diphtheria, is the blood so deteriorated in so short a time; the patient shows *grave anæmia* during convalescence.

Two *variations* of the above symptom-group are met with clinically. In *subacute rheumatism* all the symptoms are milder, and may drag on for months. "Growing pains" may be the only symptom complained of by the child, and the cardiac infection may not reveal itself till years later. *Malignant rheumatia fever* is a very serious form in which the heart is mainly involved, the joints little if at all. An eruption something like typhus may appear, and after a few days the temperature rapidly rises and the patient dies.

The *Diagnosis* of rheumatic fever in the adult is not as a rule difficult. Acute gout is distinguished by its sudden onset, and by the other features mentioned in the table given above. Acute rheumatoid arthritis affects chiefly the larger joints of the fingers (§ 469). The swelling is fusiform, and does not subside under treatment by salicylates. Pyæmia (when arising from some internal cause) may closely resemble rheumatic fever, but in pyæmia the joint inflammation is not erratic, the pyrexia is typical, accompanied by rigors, cerebral symptoms, and an enlarged spleen. Gonorrhœal arthritis usually affects the knees or the small tarsal or carpal

joints. The condition is more chronic, and there is a history of gleet. Among the other diseases which sometimes have to be diagnosed are dengue, which has a characteristic eruption ; trichinosis, in which the pain and swelling are referable rather to the muscles, and are preceded by gastric symptoms ; ulcerative endocarditis, in which the joint swelling is absent, and the temperature intermittent. Osteomyelitis starting near the epiphysis is a condition always to be borne in mind.

In infants, in whom it occurs rarely, its detection may be difficult, as it may closely resemble infantile scurvy, which is known, however, by the swollen gums and failure of treatment by salicylates. Syphilitic epiphysitis and arthritis are known by the great local tenderness and œdema extending beyond the joints, and the rapid improvement under mercury. Rheumatism in infancy may also be mistaken for infantile paralysis.

Prognosis.—The disease is not dangerous to life when it attacks the joints only, but if the heart is affected the prognosis is more grave. One attack predisposes to future attacks. Other untoward symptoms are hyperpyrexia and cerebral symptoms. An attack is grave in proportion to the height of the temperature, the implication of the heart, and the presence of cerebral symptoms. The latter, happily rare, are of the gravest import unless accounted for by salicylates. The visceral manifestations of rheumatic fever are more serious than the disease itself. The chief of these relate to the heart, which should in all cases be examined daily as a matter of routine. Nodules are usually, though not always, of grave import.

Etiology.—Age is the most important predisposing factor, acute rheumatism being almost confined to persons under twenty-five, the commonest age being between seven and twenty. It is comparatively rare under ten and extremely rare in advanced life. Dr. F. Langmead's investigations among school children revealed the fact that one in fifteen of those over seven years was rheumatic, and in 87 per cent. a cardiac lesion developed. Males seem slightly more prone to the disease, and heredity plays a considerable part. Among the determining causes may be mentioned exposure to cold or chill, and fatigue. Acute rheumatism is apt to follow tonsillitis, scarlatina, or chorea, or to precede the latter.

The clinical evidence of the resemblance of the symptoms of acute rheumatism to those of septic infection—especially in its involvement of the endocardium and the serous membranes—also supports the view of its being a specific infective disease. In 1900 Drs. F. J. Poynton and Alexander Paine isolated a diplococcus from the blood, exudates, and cardiac valves of rheumatic cases, which answered the tests of specificity. This work has been corroborated by others, but still awaits general acceptance. Dr. Poynton believes the infection enters by the throat.

Treatment.—Absolute rest in bed is necessary. It is a good plan to fold children in blankets. The diet is that for pyrexia (§ 421). As regards drugs, salicylate of soda or salicin, first adopted by Dr. Maclagan, is by some regarded as a specific. In most cases it abolishes pain and fever within a week, but the treatment must be continued, else the symptoms will relapse and the pain return. The drug must be given in large doses —gr. 20 (1·35) every two hours during the first day or two, then every

four or five hours, till the temperature subsides or physiological symptoms of the drug ensue—viz., headache, deafness, and buzzing in the ears, albuminuria, or delirium. Danger-signals to be looked out for as indications to omit or decrease the drug are vomiting, acetonuria, drowsiness, hæmaturia, and air-hunger—all indications of an acid intoxication.• When salicylates fail to control the fever the presence of pericarditis can often be deduced. To distinguish whether delirium is due to excess of salicylates or to the onset of pericarditis, the temperature chart is usually a sufficient guide. If initiated early and before cardiac or other complications have arisen, this treatment is certain to relieve (Fig. 120). In a few cases, however, especially, those in which the joints appear to be less involved than the heart, and those in which the temperature is very high, salicylates may fail. Then antipyrin or antifebrin are remedies of considerable value ; in any case, they relieve the pain and may be good for this purpose alone ; morphia should be avoided. Alkaline carbonates used to be given alone in large doses ; they are still given, but in combination with salicylates. Dr. Lees recommended very large doses of salicylate of soda, and avoided acid intoxication by giving twice as much sodium bicarbonate as salicylate. It is essential that the bowels should be opened before the drug is given, and at least once a day during its administration. The joints should be swathed in cotton-wool. An alkaline lotion containing opium (F. 35) may be used if any local application is necessary. If, in spite of the salicylates, the temperature remains high, quinine (10 grains every two hours till symptoms arise) may be given. Drugs failing, and the temperature being over 104·5°, a graduated hot bath should be immediately given, as the condition of the patient requires prompt and energetic measures. If heart complications arise, iodides should be given, and many recommend small blisters (the size of a florin) to be applied over the left upper chest or over the heart itself. During convalescence treatment is required to avoid relapses and second attacks. The patient should always wear flannel, avoid exposure, and be careful in his diet (see Chronic Rheumatism). When school children suffer with " growing-pains " or tonsillitis, a strict watch should be kept on them, and rest in bed ordered if the heart show any suspicious signs.

III. Acute Gonorrhœal Arthritis (Gonorrhœal Rheumatism) is an acute arthritis resembling " rheumatic fever," due to infection by the gonococcus from the urethra during the acute stage of gonorrhœa. It is more frequently met with in the chronic form described in § 472. If the disease arises in the acute stage of gonorrhœa, the joint mischief resembles acute rheumatism in all respects excepting : (1) Although the inflammation spreads from joint to joint, those first involved do not get better as the others become involved ; (2) the temperature has more of an intermittent character than ordinary acute rheumatism ; (3) it does not yield to salicylates, but runs a prolonged course of many weeks or months ; and (4) there is less tendency to heart complications. The joints rarely suppurate, but the disease is most intractable and may lead to extensive

adhesions and distortions in the various articulations. It is by no means unknown in children.

IV. **Acute Rheumatoid Arthritis** may start in a manner indistinguishable from rheumatism, but the joint swellings persist and become more typically those of rheumatoid arthritis later (§ 469).

V. **Pyæmia** has already been described in § 413. In some cases of acute general pyogenic infection the joints are not at all involved (septicæmia), but in others of a pyæmic type there is a marked tendency to a suppurative inflammation in and around the joints. It is differentiated from other joint lesions by : (1) the swelling does not shift its position, as it does in rheumatism ; (2) the joint may be red and show evidences of suppuration ; (3) the constitutional symptoms are very characteristic, especially the wide and irregular range of temperature and the rigors and sweatings ; (4) some cause may be revealed in the shape of an internal or external pyogenic focus (§ 414).

VI. Other **acute specific diseases** may—though less frequently than the foregoing—lead to inflammation of joints. The joint disease can be identified only by the presence or history of the disease which it complicates. In *adults* pneumonia and enteric fever may be complicated or followed by a suppurative affection of the joints, often with a fatal issue. More rarely other acute specific fevers are so complicated. In dengue joint swelling is often part of the disease ; in Mediterranean fever the joints are often affected. Cerebro-spinal meningitis is almost always accompanied by synovitis. In *children* it commonly follows scarlet fever, especially when there have been severe faucial symptoms. The lesion may be suppurative. Measles, enteric, mumps, and influenza are rarer causes in children. Synovitis sometimes follows the administration of antitoxins (§ 485).

VII. There are three remaining generalised disorders associated with joint trouble—viz., Purpura Rheumatica, Scurvy, and Hæmophilia.

§ 466. Purpura Rheumatica (Synonyms : Peliosis Rheumatica, Schönlein's Disease).—In this disease a synovitis resembling rheumatism is associated with a purpuric or erythematous eruption.

Symptoms.—(1) Many joints are affected with considerable pain and swelling ; (2) the temperature varies between 100° and 103° F., the pyrexia usually preceding the arthritis by a day or two, and being accompanied by more or less sore throat ; (3) the eruption, which usually starts upon the legs near the joints, is of a purpuric, urticarial, or erythematous character. It is attended by a good deal of anæmia. The patient usually recovers in the course of a few weeks, but the disease is apt to recur. The throat symptoms may lead to sloughing of the uvula. The blood change which underlies this condition is unknown.

Treatment.—Large doses of calcium chloride are useful ; the drug can be injected into the muscles (1 grain in 100 drops water) once daily, or 10 o.c. serum may be tried. Anti-rheumatic treatment is usually adopted.

HENOCH'S PURPURA resembles purpura rheumatica very closely. It is chiefly met with in children, but may also affect adults. There are recurrent attacks of slight pain and swelling of the joints, but the chief symptom is severe colic, occurring independently of diet ; there may be tenderness over the transverse colon. Vomiting may be severe, with or without blood. Blood or blood-stained mucus may be passed

frequently. Hæmaturia may occur. The skin lesions, erythema, urticaria, and purpura, may appear apart from the gastro-intestinal signs, or they may be absent. The attacks recur at intervals for years, without apparent cause. Treatment is symptomatic.

The condition is apt to be mistaken for intussusception.   •

In scurvy (§ 440) non-suppurative swellings occur beneath the periosteum near the joints, but the joints themselves are not often affected. The disease is recognised by the spongy bleeding gums, anæmia, and other symptoms of scurvy (q.v.).

In hæmophilia (§ 441) the larger joints are usually affected. The joint lesion is probably always due to the extravasation of blood or blood serum into the joint cavities, and usually supervenes suddenly on a slight blow or exposure to chill. It not infrequently recurs, and may ultimately lead to ankylosis. It is diagnosed mainly by the history of hæmorrhages in the patient. The condition is met with for the first time most often between the ages of seven and fourteen.

VIII. **Acute Traumatic Synovitis** is recognised by the history of an injury, though one must bear in mind (1) that many constitutional processes, especially gout, are lighted up by a very slight injury, and (2) that in childhood the history of a traumatism may be wanting.

IX. **Extension** from epiphysitis or osteomyelitis (§ 477) or other bone disease in childhood—set up very likely by injury—may produce acute inflammation in a joint, and the serious nature of the condition may be overlooked unless the correct meaning of the pyrexia and constitutional disturbance is appreciated.

(b) Chronic Joint Diseases

Joint disorders which may be chronic *ab initio* come clinically under eleven headings.

  I. Chronic gout.
  II. Chronic rheumatism.
  III. Rheumatoid arthritis.
  IV. Osteo-arthritis.
  V. Spondylitis deformans.
  VI. Chronic gonorrhœal arthritis.
  VII. Other forms of chronic suppurative arthritis.
  VIII. Tuberculous joint disease.
  IX. Syphilitic arthritis.   •
  X. Hysterical joint affection, which is often in reality a muscular stiffening and immobility.
  XI. Neuropathic arthritis (e.g., Tabes, Syringomyelia, and Raynaud's disease).

Clinically many of these joint diseases resemble each other very closely, both in their physical signs and their history, and many cases are met with which it is almost impossible to place definitely under one or other disease. Moreover, in their pathology we find the same resemblance, for with the possible exception of hysterical and neuropathic arthritis they are all due to a blood change of microbic or metabolic origin.

§ **467. I. Chronic Gout** usually supervenes upon a succession of acute attacks (§ 464) ; occasionally it is chronic or subacute from the beginning. The joint is stiff and painful on movement, is very tender, sometimes red, and sometimes masses of urate of soda (chalk stones) can be seen through the skin. The patient, who is usually a male over middle age, suffers also from gouty dyspepsia, irritability of temper, and frequent subacute exacerbations of joint trouble. Tophi are usually present. They consist

of nodules of sodium biurate, analogous to the deposits in the joints, and are commonly situated in the cartilage of the ear, near the helix, and in bursal sacs. The urine may contain a little albumen from time to time. The arteries are generally thick, and there is a marked tendency to high blood-pressure.

The *Diagnosis* between chronic rheumatism and chronic gout is by no means easy. In an infirmary, where a large number of both diseases in the chronic form are always to be seen, it is usually impossible to classify more than one-third of them. In general terms, chronic gout attacks the smaller joints, the patient is of a plethoric type, and there are concurrent symptoms such as tophi in the ears, interstitial nephritis, or the history of typical paroxysms, which give us some indication of gout. The serum test of acute gout is not of great assistance in the chronic disease (see also table, § 468).

The *Prognosis* of chronic gout is more serious than that of chronic rheumatism, though in both the same crippling of the joints occurs. Interstitial nephritis (granular kidney) is almost sure to supervene sooner or later, and the prognosis mainly depends on three factors : (i) the condition of the kidneys ; (ii.) the degree of blood-pressure ; and (iii) the condition of the heart, especially of the heart wall. The complications in addition to those mentioned under Acute Gout are (1) bronchitis ; (2) iritis and scleritis ; and (3) deposits of urates not only in the conjunctiva but in any other tissue of the body. Urethritis may occur in males ; stone is not uncommon, and sometimes glycosuria is seen. The patient may eventually die with uræmia, pericarditis, pleurisy, peritonitis, meningitis, or apoplexy. The *Treatment* is described under Acute Gout.

§ 468. II. **Chronic Rheumatism** is a common affection of the joints. The disease may follow one or more acute attacks, or, as is more usual, come on insidiously as a chronic affection from the beginning. Chronic rheumatism is believed to be distinct from acute rheumatism. The capsule, ligaments, and tendon sheaths are thickened. The joint is stiff and creaks with adhesions, is generally more or less swollen, and sometimes tender. Sometimes many joints are affected symmetrically ; sometimes only one. Ultimately the joint may be considerably distorted, but not disorganised, there being a tendency towards adhesions and fibrous thickenings. When the hands are affected they are in time permanently deformed. The general health, in many cases, is not disturbed, though there is a certain amount of anæmia and a variable degree of pain. The disease is never fatal.

The *Diagnosis* from chronic gout is sometimes very difficult. Many hold that it is the same disease as rheumatoid arthritis, that when chiefly the synovial membrane and periarticular structures are involved, the malady is labelled chronic rheumatism, and when degenerative changes occur in cartilage and bone it is called rheumatoid arthritis or osteoarthritis.

Apart from arthritic heredity the *Etiology* is obscure. The patient is

TABLE XXVII.—TABLE OF DIAGNOSIS.

Chronic Rheumatism	Chronic Gout	Rheumatoid Arthritis.	Osteo-arthritis
Either sex middle life or over	Generally male sex, over forty.	Chiefly female sex, usually twenty to forty.	Females more than males, forty to sixty
Poor and debilitated Insidious onset, unless following rheumatic fever	Rich and plethoric generally History of sudden onset and acute attacks with severe pain Skin over joints red, swollen, and œdematous	More common in the poor Onset acute, subacute, or insidious Constitutional symptoms present	More common in poor and debilitated. Onset, insidious, course, progressive No constitutional symptoms
Generally polyarticular • Temporo maxillary joint not affected	Only one joint affected at first usually the metatarsophalangeal of the great toe	Generally polyarticular Temporo maxillary joint often affected Spreads from the smaller joints to the larger, terminal interphalangeal joints usually unaffected	Polyarticular or monoarticular Temporo maxillary joint affected terminal interphalangeal joints usually affected
Thickening of tendons and ligaments, no bone changes	Deposits of urate of soda round the joints	Spindle-shaped enlargement with ulnar deviation and later some fixation No lipping or osteophytes	Radial deviation of terminal phalanges lipping and osteophytes marked

generally past middle life, unless chronic rheumatism has followed acute attacks, when the patient may be younger.

Treatment is discussed under the treatment of arthritis (§ 470).

§ 469. III. Rheumatoid Arthritis. —The terms Rheumatoid Arthritis, Osteo-arthritis, Rheumatic Gout, and Arthritis Deformans have been long used as synonyms, but some hold that the first two are separate clinical entities and that rheumatic gout is an inaccurate term.

Rheumatoid arthritis is a general disease, producing synovitis and peri-arthritis, as shown by swelling and pain in the joints. It tends to get well, often after a protracted course, but leaves, not uncommonly, considerable deformity and crippling. It manifests also symptoms of constitutional disturbance.

Symptoms.—The onset may be acute, subacute, or chronic. In the first form the condition closely resembles acute rheumatism at first, but the joints prove intractable to the action of salicylates, and later assume the typical characters. In the subacute variety the joints are rapidly affected, but show only slight swelling at first, whilst the temperature is but little raised. The chronic form begins insidiously in one joint, and spreads slowly.

1. The joints usually affected first are the proximal row of the interphalangeal joints of the fingers, and the metacarpo-phalangeal joints, next the wrists, ankles, and knees; then the shoulders, and last of all the hips, so that the progression of the disease in the joints is from the

periphery. The lesions are symmetrical. The temporo-maxillary and vertebral joints are specially liable to be attacked. The distal interphalangeal joints are usually spared. During the active stage the joints are painful, tender, and swollen, and somewhat limited in movement. The swelling is fusiform, due to the fact that the lesion is a combination of synovitis and peri-arthritis; and there are neither lipping of bone or osteophytes to be felt, nor can grating be elicited. If the active stage is of long duration, this may be followed later by very marked limitation of movement and deformity, due to the formation of adhesions within and around the joint, and to the secondary contraction of muscles. In severe cases partial dislocation or ankylosis may occur. The most common displacement is that of ulnar deviation of the fingers (Fig. 121). The muscles above and below the affected joints are conspicuously atrophied, to a much greater extent than could be explained by disuse. The tendon reflexes are increased.

Fig. 121.—RHEUMATOID ARTHRITIS in a man about thirty-eight years of age, showing ulnar deflection of fingers.

2. Subcutaneous nodules are sometimes present. Usually these are in the form of flat masses in bursæ, especially the olecranon bursa ; but more rarely they resemble the nodules of rheumatism, differing from them in being more permanent and occasionally tender.

3. The skin is glossy, atrophic, and apt to become parchment-like on the backs of the hands and fingers, which are often cold. Pigmentation is common, and may occur as circumscribed spots like freckles, or as diffuse spreading patches. It occurs especially on the face and neck, and on the backs of the wrists and forearms, but may be general. The forehead may shine like burnished bronze and various tints of yellow and brown are seen by reflected light at different angles. A brawny œdema of feet and legs may be present, independent of cardiac or renal disease.

4. The axillary and inguinal glands are not uncommonly swollen.

5. Constitutional symptoms. There is usually some fever during the active stage, the temperature varying from normal to as high as 102° or 103° F. The pulse is nearly always quickened, and may be from 90 to

100 for years. The general nutrition is impaired, and the patient is usually pale and depressed.

The *Diagnosis* is considered in the table on p. 665.   •

In *children* certain forms of arthritis occur which resemble the lesions of rheumatoid arthritis in adults. The multiple arthritis described by Dr. G. F. Still (Still's disease), associated with pallor, fever, wasting, and enlargement of the lymphatic glands and spleen, differs from the description given above only in the frequency of affection of the glands and the splenic enlargement. It is probably rheumatoid arthritis modified by the age at which it occurs.

Prognosis.—The prognosis of all forms of peri-arthritis and even of rheumatoid and osteo-arthritis has become more hopeful of recent years. This is especially true if the case is seen early and an infecting focus can be traced and removed. When the glucose tolerance is greatly diminished and the source of infection cannot be removed, the outlook is serious; this often occurs when the mucous membrane of the intestine is involved. The course is slow, with many relapses, and there is much crippling and deformity. The disease is rarely fatal.

Etiology.—The disease may occur at any age, but is most common between twenty and forty. Females are more often affected than males, in the proportion of about three to one. A lowered resistance frequently precedes an attack, which may in this way follow acute infections, especially influenza, or overwork and anxiety. Apical abscesses, pyorrhœa alveolaris, septic conditions of the nose, antrum, sinuses, ear and throat, tonsils, pelvic disease, cystitis, colitis, and ulcerating piles are among the conditions which act as infecting foci. Sometimes the infecting cause lies in the gall-bladder or the digestive tract; sometimes it cannot be traced.

Treatment is discussed in § 470.

§ 470. IV. Osteo-arthritis is a chronic degenerative disease of joints, progressive in character, and occurring chiefly in the elderly.

Symptoms.—The special features of the joints are as follows : The ends of the bones are thickened and lipped. The synovial membrane is also thickened, and thickened fringes can be felt, in some of which cartilaginous bodies are recognisable. These may be pedunculated or free, forming the so-called melon seed bodies. Bony outgrowths or osteophytes are formed, often in great quantity, so that if the joint is moved, scrunching or grating is audible, and by their interlocking, movement is much restricted. True ankylosis rarely occurs. The joint is often distended with fluid, as are also the bursæ, around it. Sometimes the encysted collections of fluid near the joint are unconnected with bursæ, but are lying in spaces bounded by muscles and areolar tissue. Pain is not usually severe, but the joints often feel hot and tingling, and occasionally numb. In severe cases considerable deformity results from absorption of the ends of the bones, so that shortening or displacement is produced. There is no constitutional disturbance in this disease. Muscular atrophy occurs, but is less marked than in rheumatoid arthritis, and does not lead to the same crippling by contracture.

The disease may be localised or general:

1. *Heberden's Nodes* form the commonest and best known variety. These are bony growths, which occur at the sides of the distal interphalan-'geal joints. They are usually painless, but may be painful, and produce numbness and tingling in the fingers. Little bursal swellings occasionally accompany them. In advanced cases the terminal phalanges are bent acutely toward the radial side. The hands are symmetrically affected. This condition may exist alone as evidence of osteo-arthritis, but often accompanies other varieties.

2. *The Carpo-metacarpal Joints of the Thumbs* are not infrequently affected alone, or with Heberden's nodes. The joints are loose and grate, and the bones can be felt to be lipped.

3. *The Knees* are frequently affected in women at the menopause. Pain and stiffness is noticed on walking or going downstairs, and the knees give way, letting the patient down. If the joint be moved when the patella is depressed by pressure with the thumbs, a fine velvety scrunch can be felt and heard. Later the joint assumes the ordinary deformity.

4 *The Temporo-maxillary Joint* may be first or solely affected, and the osseous outgrowths may lead to locking so that chewing is impossible. This joint is not so often affected as in rheumatoid arthritis. *

5. *The Hip-Joint of Elderly Men.*—This is the most important local form of the disease, since it leads to considerable crippling. It is usually unilateral. There are pain and rigidity of one hip-joint with difficulty in adduction. The pain is felt most severely in the groin, but may radiate down the front of the thigh to the knee. From sciatica, with which it is often confused, it is distinguished by the position of the pain, and the fixity of the joint. Wasting may occur later, but is limited to the buttock and thigh. The limb may be shortened. It occurs chiefly in men over fifty.

6. *The Generalised Form.*—In this condition most of the joints in the body may be attacked, including those of the spine. In the hands, the distal interphalangeal joints and the carpo-metacarpal joints of the thumbs are usually selected, and show the characteristic grating and lipping, not the fusiform swelling of rheumatoid arthritis.

Prognosis.—If treated early, temporary improvement may occur, but speaking broadly, the disease is progressive. The form occurring in the hip-joint of old men is very intractable, but that in the knees of women at the menopause more remediable. The crippling is not great, but patients with the joints of the lower extremities affected will often be afraid to get about, because of the fear of the knees giving way. ,

Etiology.—It occurs most often in women between forty and sixty years of age. It is doubtful whether the joint lesions are in any way specific, since similar changes occur as the result of traumatism of prolonged pressure, as by a tight boot, in hæmophilia from repeated hæmorrhages, and in tabes dorsalis and syringomyelia.

' *Treatment.*—The treatment of all forms of arthritis has made great

advance in recent years. During the active stage rest is essential, and the joint when at rest must be maintained in a good position. When pain has diminished, massage and weak doses of galvanism are indicated to prevent wasting and fixation. The diet should be nourishing and abundant, consisting of milk, eggs, green vegetables, meat and plenty of fats. The sugar tolerance of arthritics is lowered [1]; therefore carbohydrates must be reduced and sugar and sweet wines forbidden. Possibly this explains the success of the once famous Salisbury diet, which consists chiefly of meat. The infecting focus must be carefully sought for and removed. The teeth have often been needlessly extracted, but where extensive pyorrhœa and apical abscesses are present extraction is necessary. Endocervicitis is a frequently overlooked focus of infection. Autogenous vaccines give good results when skilfully administered over a prolonged period. Start with small doses of under one million, increasing very cautiously and avoiding all reactions. Reactions from overdosage throw back progress and cause unnecessary suffering. Intravenous protein therapy is much advocated by some for multiple peri-arthritis, and has done good for some cases of rheumatoid and osteo-arthritis. It is contra-indicated in the old and feeble. Six injections are given, every fifth day, of typhoid or B. coli (30 worked up to 200 millions), or Armour's peptone. Each dose is followed by a rigor and temperature, and later, much improvement. Drugs are very useful in most cases. Tincture of guaiacum, half-teaspoonful doses in milk thrice daily, has proved useful. Potassium iodide, or French iodine (which contains no potassium) can be continued over a long period. It is given in 1 drop doses in milk, thrice daily, cautiously increased to 30 or 40 drops, then decreased and again increased. Other useful preparations of organic remedies are iodine, guaiacol carbonate, collosol sulphur or iodine, arsenic and iodide of iron. Paraffin, with kaolin or other intestinal antiseptics, suits certain cases; in others, hydrochloric acid is indicated, to combat the deficient secretion; in others, thyroid or parathyroid do good.' As regards climate, dry warm places such as Egypt are most beneficial. Possibly it is the sunshine which is so efficacious; certainly heliotherapy benefits even when the infecting focus has not been found and removed. Spa treatment is obtained at Bath, Buxton, Harrogate, Aix-les-Bains, etc. There many forms of treatment by heat are administered. With the Tallerman bath, the joint is placed for twenty minutes in a copper chamber at a temperature of 250° or 300° F. Radiant heat baths do so much good to chronic stiffened joints that it is probable the benefit is due to the light as much as to the heat. The pyretic couch treatment has also many advocates. Dr. Luff's iodine vapour bath relieves. The joint is washed and dried, the skin painted with tincture of iodine, and covered with a thin layer of butter muslin. A linseed poultice is placed over this, and the part smothered in cotton-wool. Other local applications are Scott's dressing,

[1] Pemberton: Arch. Inter. Med. 1920; Kerr Pringle and Miller; The Lancet, Jan., 1923.

guaiacol, linimentum potassii iodidi cum sapone, and equal parts of guaia-
col, menthol and linimentum camphorse. Ionisation and diathermy are
excellent in some cases.

§ 471. V. Spondylitis Deformans is a disease formerly classed under rheumatoid
arthritis, but now recognised as a morbid entity. The vertebral column and the
shoulder and hip joints are most often affected.

⮞ *Symptoms.*—The spine may be quite rigid, so that the name " poker back " is
aptly applied. This is due to a synostosis of the vertebræ and ossification of the
intervertebral ligaments. A similar change at the hips and shoulders may produce
fixation, partial or complete, of these joints also. There is marked kyphosis of the
upper part of the spine. The chest is flattened, and the breathing is sometimes
entirely abdominal, due to fixation of the costovertebral joints. Nipping of the
nerves at their exit between the vertebræ may lead to referred pains around the chest
or abdomen, areas of impaired sensation, paræsthesia, and local atrophy of muscle.

Etiology.—It occurs chiefly in adult males, but has been described in children.
Three children in one family have been attacked. Gonorrhœa is now suggested as
the cause.

Treatment is symptomatic, and on the same lines as that for osteo-arthritis.

§ 472. VI. Gonorrhœal Rheumatism (Synonyms : Gonorrhœal Arth-
ritis, Urethral Arthritis) is a synovitis following a gonorrhœal discharge
resembling chronic rheumatism in some respects, chronic pyæmia in others.
An acute form has been referred to on p. 661, but the disease is nearly
always chronic. In this, the chronic and commoner form, the joint affec-
tion comes on insidiously during the gleet (often about the fourth or fifth
week). According to surgeons who see many of these cases, it is particu-
larly apt to supervene in those cases of gonorrhœa in which the prostatic
portion of the urethra is affected, and the extreme vascularity of that part
lends probability to this view. When the joint becomes involved, the
gleet sometimes disappears, a circumstance which may give rise to an
error in diagnosis. In the chronic variety there is only slight elevation
of temperature of an intermittent or hectic kind ; but the general health
is always more or less disturbed, and may be so greatly that the patient
becomes anæmic and emaciated. The disease attacks many of the joints
usually spared in other diseases, such as the sacro-iliac, sterno-clavicular,
and temporo-maxillary, and is apt to settle down in the smaller joints
of the carpus or tarsus. The affected joint becomes swollen, stiff, and
tender, and gradually ankylosed. The fibrous tissues also are often affected,
especially the plantar fascia ; pain in this position or in the tendo Achilles
is an important diagnostic feature of the disease.

For the *Diagnosis* one has to rely mainly on the history of gonorrhœa,
the inveterate character, the tendency to ankylosis, and the fact that it
is wholly unrelieved by salicylates.

The *Prognosis* as regards life is favourable. It is more hopeful in younger
people and in attacks of recent date. The heart is seldom affected, but
there may be pleurisy or iritis, and in rare cases the meninges have become
affected, with fatal result. The probability of cure depends on the cura-
bility of the urethritis, which with modern methods is more

As regards *Etiology*, both men and women may be affected. It has been definitely shown that the gonococcus may be present in the joint. Special exposure to chill during a gonorrhœa will sometimes determine the disease.

Treatment.—The first indication is to cure the urethritis. This is sometimes extremely difficult. Hexamine, helmitol, cystopurin and other urinary antiseptics may be tried. Local applications to the urethra consist of silver nitrate, argyrol, protargol, and potassium permanganate. Ionisation and diathermy of the female urethra yield a prompt cure. For the joint mischief one of the most useful applications is Scott's dressing with ung. hydrargyri or oleate of mercury, but of recent years local diathermy has superseded all other forms of treatment. The gonococcus cannot exist at the temperature to which the joint is raised.[1] It is of the greatest importance to improve the patient's general nutrition. The patient should avoid thereafter any possibility of a fresh attack of gonorrhœa. Gonococcus vaccine has yielded good results, and should be tried.

VII. Other forms of **Chronic Septic Arthritis.**—Chronic infective arthritis, a rare form of chronic joint affection. A case which I had under my care for many months is described and illustrated in § 414. The joint mischief was probably due to secondary infection from an internal pyogenic focus—namely, in the appendix. Cases of arthritis have occurred during convalescence from dysentery (see § 244).

§ 473. Tuberculosis, Syphilis, Hysteria, Tabes Dorsalis, and other nervous disorders also affect the joints.

VIII. **Tuberculous Joint Disease.**—Tuberculosis affects chiefly the synovial membrane, but it may commence in the articular ends of the bones. This is *par excellence* the monarticular joint disease of children.

Symptoms.—The onset is insidious, though not infrequently the symptoms date, or are supposed to date, from an injury. The favourite situations are hip and knee-joint, though any joint may be affected. The child may complain of slight pain, which gives rise to limping, for weeks or months before anything is apparent. Generally the disease is in the knee, but sometimes it is in the hip, although the pain may still be referred to the knee, one nerve supply of which is also a branch of the obturator nerve. By and by the affected joint swells; it is pale, and has a pulpy or doughy feel beneath the finger, and fluctuation may be felt. If untreated, the case goes on to abscess formation. The constitutional symptoms consist of an intermitting pyrexia, and general debility which are present even from the very beginning.

The *Causes* are the same as those mentioned under phthisis. The symptoms may date from or be first noted after an injury. The disease nearly always attacks children, though a more destructive form of tuberculous joint mischief does occur in advanced life. It may last for many years, and the prospect of recovery depends very much upon the stage at which it first comes under treatment. If neglected, extensive destruction of the joint may occur, and very frequently tuberculous mischief is found in other organs. The *Treatment* is mainly dealt with by the surgeon, but a good deal can be done in the early stages by rest, fresh air, and cod-liver oil.

IX. **Syphilitic Joint Disease.**—In the secondary stage of syphilis there may be (i.) a subacute arthritis with redness and pain, or (ii.) an indolent hydrarthrosis, with

[1] *Diathermy,* by Elkin Cumberbatch.

little pain. In the tertiary stage of syphilis the differential features of the arthritis are :—(1) One or several joints may be affected. The synovial membrane may be attacked, leading to a doughy swelling; or the ligaments or cartilage. (2) The joint manifests no signs of acute inflammation, but there is occasionally some effusion. (3) The pain is very moderate during the day, but subject to nocturnal exacerbations. (4) Other evidences of syphilis are generally present. (5) The condition is very chronic, and is only partially amenable to iodides. It may occur in children.

A PSEUDO-PARALYSIS OF SYPHILITIC ORIGIN occurs in infants, due to the separation of the cartilage from the diaphysis, and is apt to be mistaken either for joint disease or for infantile paralysis. The affected part is, however, acutely tender.

X. **Hysterical Joint Disorder** usually affects the hip or the knee, and it often dates from some trifling injury. The joint is fixed, tender (often more tender to light touches than to deep pressure), and sometimes swollen, and the local temperature of the joint may also be raised. Sometimes there are no physical signs referable to the joint at all. The loss of function may be entirely due to muscular rigidity, and in the case of the hip-joint the condition may very precisely resemble (mimic, as Sir Jas. Paget says) tuberculous disease of this joint. This *Diagnosis*, which is often extremely difficult, rests mainly on (1) the absence of evidence of serious disease in the affected joint when examined under chloroform ; (2) the disproportionate loss of function ; (3) the patient being a female, and the subject of other manifestations of the hysterical diathesis.

The *Treatment* should be mainly directed to the hysteria (q.v.). The joint mischief may sometimes be cured by chloroform anæsthesia ; on coming round the patient finds that she can use the joint, and continues to do so. In the author's view these cases are due to a vascular change in the synovial membrane, probably of vaso-motor or toxic origin, albeit slight, and perhaps temporary and evanescent. Many cases of undoubted hysterical joint disease are amenable to salicylates, or alkaline carbonates on the one hand, or to bromides or vaso-motor remedies on the other.

XI. **Neuro-trophic Arthritis** (Synonyms : Neuro-Arthropathy, Tabetic Arthro-pathy, Arthritis in connection with spinal lesions).—Two diseases of the spinal cord are sometimes, though comparatively rarely, attended with chronic mischief in the joints —viz., *Tabes Dorsalis* and *Syringomyelia*. In both it may occur in an early stage of the disease, when nervous symptoms are few or absent, and in both extensive disintegration of the joint may take place, without pain, heat, or redness, and without giving rise to much inconvenience. In *tabes dorsalis* the associated joint lesion is known as tabetic arthropathy, or Charcot's joint disease, because it was he who first identified the connection. This lesion may occur without the patient suffering any pain, and but little inconvenience, although the bone ends may be enlarged, and it may go on to extensive disorganisation with increased mobility and new bony forma-tions before the patient seeks a doctor's advice. A case of *tabes dorsalis* is narrated by Prof. J. M. Charcot of a soldier, in whom actual dislocation of both hips was found to have occurred without the patient being aware of any mischief in the joints. Indeed, it had happened when he was on the march. In all such cases the pupils and knee-jerks should be examined. The knee is the favourite situation, and therefore it is sometimes difficult to test the tendon reflexes.

Syringomyelia is characterised by muscular atrophy and anæsthesia at the ends of the extremities. Any joint may be involved ; in two cases I have seen very extensive disease affected the joints of the upper extremities.

In *Raynaud's Disease* a subacute or chronic synovitis sometimes occurs which is possibly of vaso-motor origin.

Intermittent Hydrarthrosis possibly comes under this heading. The joints swell at periodic intervals which the patient can foretell almost to a day. Nothing is known of its etiology. It has been known to alternate with attacks of psoriasis, and is perhaps symptomatic of some inborn error of metabolism.

GROUP III. MUSCULAR DISEASES

We are here concerned with lesions situated in the muscular substance as evidenced by pain in the muscle (myalgia) and tenderness, accompanied, perhaps, by some swelling. The causes of pain in the limbs were discussed in § 452. The causes of muscular weakness will be dealt with in the chapter on nervous diseases.

I. Muscular rheumatism or gout.
II. Tumours.
III. Trichinosis.
IV. Idiopathic myositis.

§ 474. I. **Muscular Rheumatism** (fibrositis) is certainly the most frequent cause of muscular pain and tenderness in this country. It is difficult, if not impossible, to separate gouty from rheumatic muscular inflammation.

Symptoms.—(1) The pain usually comes on quite suddenly; so suddenly indeed, in the case of lumbago, that it is often mistaken for a sprain or rupture of the muscular fibre. It is greatly aggravated by movement and relieved by rest. In the more acute cases it is attended by localised tenderness. (2) Very little swelling can be detected in the affected muscles, a point of distinction from trichinosis and new growths. (3) It is usually accompanied by a furred tongue and disordered digestion, with constipation and a copious deposit of urates in the urine. There may be slight pyrexia.

The commonest *variety* of muscular rheumatism is **lumbago,** where the pain is situated in the muscles and fascia of the small of the back. It is usually of very sudden onset, often when in the act of stooping. Rheumatic torticollis is a rheumatic affection of the sterno-mastoid, and is met with chiefly in children. Intercostal rheumatism is a similar affection of the intercostal muscles. Lumbago has to be *diagnosed* from other causes of lumbar pain (§§ 295 and 367). In aneurysm of the dorsal aorta the pain is more continuous, not so easily relieved by muscular rest. In myelitis and meningitis there are other symptoms referable to the nerve trunks, sensory, or motor. Muscular rheumatism, though not lethal, is very painful and incapacitating, and is very prone to recur.

Etiology.—Muscular rheumatism generally arises in gouty and rheumatic subjects who present other evidences of fibrositis. It is usually determined either by a chill, especially after prolonged exertion accompanied by profuse perspiration, or a muscular strain. It comes on especially in cold and damp weather. Cold or damp alone do not seem able to produce it; it is when the two occur together, and especially when combined with errors of diet, that the disease is found. Sugar, rich foods, and sweet heavy wines are most potent for evil. Fibrous nodules can often be felt in the muscles, tendons, fascia, and nerves in chronic rheumatism; they swell and become more painful in cold, damp weather. These are infected areas with surrounding reaction, due in most cases to extension from some

infective focus; In some cases, after massage of the affected area, micro-organisms have been found in the urine.

Treatment.—The treatment must be directed on the lines laid down for gout and rheumatism in other parts, and consists mainly of free purgation with cálomel and salines, of alkalies, sodium salicylate, or guaiacum (F. 96) ; quinine and iodide of potassium are useful in protracted cases. Rest is necessary for the pain ; and even morphia may be required. Locally, counter-irritants are best. Lint soaked in a mixture of equal parts of liniment of belladonna and chloroform should be kept over the muscles (or in hospitals, liniment of turpentine does equally well, and is .very much cheaper) ; it should not be covered up with oil silk, or it will blister. Radiant heat is very efficacious. The galvanic current allays the pain and promotes recovery in some cases. Warm underclothing and a flannel belt may prevent recurrence. The diet must be simple ; sugar and alcohol should be avoided (see also § 468), and carbohydrates limited, because the glucose tolerance in these cases is low. Sometimes an attack may be aborted by massage and a Turkish bath. The nodules may be massaged perseveringly, in spite of the pain caused, for six or eight weeks ; this should be followed by exercises to stretch the muscles and aponeuroses. Obdurate cases often yield to an autogenous vaccine prepared from the infecting focus. Begin with small doses, 1 million, and increase slowly, avoiding all reaction ; the injections can be continued for a year or longer till high doses of 1 or 2,000 million are reached.

II. **Tumours in the substance of the muscles** may give rise to pain and tenderness, usually associated with swelling. The pain and tenderness are in this case strictly localised, at any rate at first, to the seat of the disease, and there is a thickening or tumour discoverable on careful palpation. In some cases—*e.g.*, syphilitic and malignant growths, the lymphatic glands in the neighbourhood are enlarged. The chief tumours affecting muscles are (*a*) innocent—syphilitic gumma ; abscess, which may arise from a gumma, or be of inflammatory origin ; innocent neoplasms such as fibroma, lipoma, angioma, and hydatid or cysticercous cysts. (*b*) Malignant growths, sarcoma, and carcinoma (by extension). First determine whether the swelling is inflammatory or non-inflammatory, malignant (and rapidly growing) or non-malignant, by an investigation of the swelling, the glands, the history, and the concurrent symptoms. The diagnosis and treatment is mainly surgical.

§ **475.** III. **Trichinosis** is a disease due to the presence of a nematode worm (the trichina spiralis) in the intestinal canal, and the dissemination of the embryos in the blood and the muscular system, consequent on the ingestion of " measly " meat (usually pork) insufficiently cooked. It is rarely met with now. The female *adult* or intestinal worm measures about ¼ inch, the male slightly less. In fæcal examinations for the parasite it should be remembered that the characteristic feature is the " cell body " at the anterior part of the intestine of the parasite. The *larvæ* (Fig. 122) or muscle trichinæ are found in infected muscle, where they are visible by the aid of a ¼ or 1 inch lens. Each consists of an ovoid capsule (translucent, or infiltrated with lime salts, according to the length of time it has existed) containing two or more embryos coiled up within it. The *embryos* are 0·6 to 1 mm. long, with pointed head and rounded tail. The presence of these larvæ gives to pork or other infected meat a characteristic " measly " appearance visible to the naked eye. Trichina is chiefly conveyed to man by " measly " pork, insufficiently cooked ; the capsules are digested, and the embryos set free in the intestinal canal. During the ensuing week the embryos

attain sexual maturity; each female can produce several hundred embryos. After fecundation the female worm penetrates the walls of the intestinal canal; hundreds of embryos reach the lymph spaces, blood and muscles, where after two or three weeks they become encysted. They have been found alive and capable of developing ten years after their entrance.

Symptoms.—The disease runs a course of several weeks, and shows three stages. The *first* stage lasts about a week or ten days, during which the symptoms are those of gastro-intestinal disturbance, abdominal pain with diarrhœa and vomiting. The *second* stage, which lasts from two to three weeks, coincides with the migration of the embryos. This gives rise to acute universal muscular pain, tenderness, and sometimes swelling. The wandering of the embryos in the muscles produces shortening and rigidity; the biceps seem specially apt to be affected, resulting in a typical flexion. In severe cases, movement of the affected muscles—*e.g.*, turning the eyeball, chewing, swallowing, etc., aggravates the pain. Pyrexia, of a remitting or intermitting type, is present, with profuse perspiration, inability to sleep, and possible delirium. Extreme dyspnœa arises if the diaphragm is implicated. There may be general œdema, starting in the face, and later on emaciation. In slight cases the muscular and other symptoms are insignificant and overlooked. In the *third* stage the acute symptoms gradually subside, great muscular weakness ensues, and recovery is slow. This stage is apt to be interrupted by various complications, especially pneumonia, pleurisy, or persistent and intractable diarrhœa.

In slighter cases the *Diagnosis* from muscular rheumatism or other diseases in this group may present difficulty, though the widespread muscular tenderness of trichinosis, the history of gastro-intestinal symptoms, and the epidemic occurrence in a whole family should aid us. The stools after a large dose of calomel may be searched for the adult worm, and it has been suggested to remove a small portion of muscle by means of a harpoon for microscopic examination. Some cases are mistaken for typhoid

FIG. 122.—Larvæ of the TRICHINA SPIRALIS encysted in muscle.

fever, and *vice versa.* In trichinosis there is marked leucocytosis—reaching 30,000 per c.mm. or more—due mainly to an enormous increase in the eosinophil cells which may amount to 50 per cent. of all the leucocytes as compared with the normal 2 to 3 per cent., and the embryos may be found in the blood.

Prognosis.—The disease not infrequently ends fatally between the third and sixth week; the mortality varying from 2 to 30 per cent. The intensity and duration of the symptoms is a fair measure of the prospect of recovery. Death may occur from (i.) diarrhœa, (ii.) asphyxia (from involvement of the respiratory muscle); (iii.) from exhaustion; or (iv.) from hæmoptysis or pneumonia. In any case health may not be restored for several months.

Etiology.—The disease is due to the ingestion of " measly pork " or other meat, and occurs in an epidemic form in families and towns. It is much more frequent in Northern Germany, where underdone pork or ham is a popular food, than in England and France. Thorough cooking will destroy the parasite, but in large joints the temperature may not be sufficiently high to destroy the parasite in the interior. All meat, particularly sausages and pork, should be thoroughly well cooked. It is said that 170° F. will destroy the larvæ but that 107° F. will do if maintained long enough. This temperature must permeate to the interior of the meat.

Treatment.—If the patient is seen within two or three hours after eating infected meat an emetic should be given. If the disease is discovered within twenty-four or
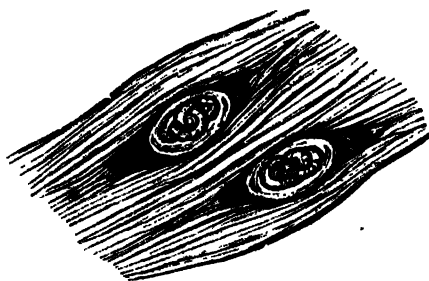
thirty hours the gastro-intestinal tract must be thoroughly cleared out. Glycerine in large doses has been recommended in the first stage for its hygroscopic properties to destroy the nematode. Filix mas, kamala, santonin, thymol, and turpentine are also recommended. A cure has been reported from the use of salvarsan. If, however, the second stage is reached, and the embryos are migrating, the treatment must be symptomatic, because nothing will destroy them. For the pain and tenderness, opium and other anodynes may be required.

§ 476. IV. **Myositis**, or inflammation and swelling of the voluntary muscles, is a rare condition; only a few cases have been placed on record. Three forms are recognised by authors : (a) A *localised* form, in which pain, tenderness, swelling, and impaired movement are localised to one muscle or group of muscles ; (b) an *acute generalised* form in which these symptoms, accompanied perhaps by œdema and redness of the skin, are more widespread ; (c) a *progressive generalised* form of myositis in which the disease runs a prolonged course spreading from muscle to muscle. An example of this variety is reported by Sir J. K. Fowler. The disorder began in the left thigh, and in about two years became general. It was characterised by painful knotty swellings, and followed by general wasting of the muscles. In *myositis ossificans* the process goes on to the formation of bone.

The *Diagnosis* of myositis appears to offer some difficulty, especially from trichi-. nosis. A microscopic examination by means of a harpoon would afford aid in the acuter cases, and the prolonged progressive course of myositis aids the diagnosis of chronic cases.

The *Causes* of idiopathic myositis are very obscure. It seems probable that Syphilis may have been in operation in some cases. Glanders and Actinomycosis also affect the muscles. The acute localised form is generally the result of injury, or spread from surrounding structures. Some think that the generalised varieties are essentially of nervous origin.

In regard to *Treatment*, iodides may be tried. Warm baths relieve the pain and stiffness.

GROUP IV. BONE DISEASES

It would be out of place here to discuss at length the diseases of the bones, which belong in a special manner to the surgeon. Nevertheless, these diseases frequently come under the notice of the physician, especially in their early stages. Pain and deep-seated tenderness are often their chief and sometimes their only symptom. Pyrexia and constitutional derangement may be present. Deep-seated swelling and deformity may appear later, and, if the bone is superficial, œdema and redness of the skin. The majority of bone diseases are chronic.

Acute Bone Diseases.

I. Acute osteomyelitis.   III. Acute epiphysitis.
II. Acute periostitis.

Chronic Bone Diseases and Deformities.

I. Rickets.   VII. Osteitis deformans.
II. Chronic periostitis, osteitis, caries,   VIII. Mollities ossium.
    and necrosis.   IX. Leontiasis ossea.
III. Tumours of bone.   X. Multiple myeloma.
IV. Acromegaly.   XI. Osteogenesis imperfecta.
V. Achondroplasia.   XII. Cleido-cranio-dysostosis.
VI. Pulmonary osteo-arthropathy.   XIII. Brittle bones.

§ 477. Acute Infective Osteomyelitis (Synonyms: Acute Periostitis, Acute Necrosis). —This disease, which used to be known as acute necrosis, and later as acute periostitis, is an acute inflammation affecting one or more of the bones, accompanied by severe constitutional disturbance—on which account the case comes under the notice of a physician. This is the only really acute bone disease, though acute symptoms very closely resembling those of osteomyelitis may arise in association with a LOCALISED PERIOSTITIS such as results (especially in children) from an injury.

The *Symptoms* of acute osteomyelitis are (1) pain of a very severe character coming on suddenly, and attended by extreme tenderness, starting usually at the articular end of the bone—very often the tibia—attended in the course of a day or two by swelling of the limb, at first pale, and afterwards red, as the inflammation makes its way towards the surface. (2) The constitutional symptoms come on suddenly, and are very marked. The temperature is high, and there are rigors and great prostration. The *Diagnosis* from acute rheumatism, which it may at first resemble, because of the pain starting near a joint, is made by the fact that in rheumatism the pain and swelling are confined to the joint, by the early involvement of other joints and by signs of cardiac complications. Acute epiphysitis is mentioned below. The *Prognosis* is always very grave. If the patient survive the initial constitutional disturbance the malady leads to abscess, necrosis and tardy convalescence. The most frequent complications are pyæmia and the extension of the inflammation to a joint.

Etiology.—Acute osteomyelitis is more frequent in children under the age of puberty. It may occur as the sequel to the continued fevers. *Treatment.* -A surgeon should be called in at once to consider the advisability of free incision, drainage, and other surgical measures.

Acute Localised Periostitis may arise from traumatism, and if not infected it soon subsides. If infected either from a wound or from the blood, suppuration and necrosis take place, and the condition becomes chronic (§ 479), which is more common than acute periostitis.

Epiphysitis is inflammation beginning in the growing line, which in early life separates the epiphysis from the shaft of the long bone. The *acute* form is met with in very early infancy. Suppuration soon sets in, and spreads to the joint, forming abscesses. It may resemble acute osteomyelitis, but the profound constitutional disturbance is lacking. It is distinguished from acute rheumatism by the age of the patient, and by the development of abscess. In the *chronic* form the process is much slower, and is of interest chiefly in relation to the diagnosis of rickets, from which it differs in being localised to one joint. As regards causation the acute form is generally due to an injury and sepsis; the chronic form is generally associated with syphilis or tubercle.

§ 478. I. Rickets (Synonym: Rachitis) is a constitutional disorder of childhood attended with epiphyseal enlargements and other deformities of the skeleton. It was described by Glisson in 1675.

The *Symptoms* for which we are consulted, coming on between the sixth and twelfth month, are delayed dentition and walking, or the child cannot sit up; gastro-intestinal disorders; bronchitis; sweating about the head; or a generalised tenderness and restlessness. In the limbs the disease is typically shown by the *enlarged epiphyses*, affecting most, if not all, of the long bones. The rib-ends are the first to show the enlargements at their junctions with the costal cartilages and thus produce an appearance of " beading "—the " rickety rosary." The long bones often curve, a condition most commonly seen in the tibiæ and fibulæ. The convexity of the curve is outwards, and greenstick fractures may be produced by slight injuries. The spine has a general backward curvature when the

TABLE XXVIII.—CHRONIC BONE DISEASES AND DEFORMITIES.

	Occurrence.	Age Period most Affected.	Number of Bones Affected.
Rickets.	Very common.	Infancy (six months to two years).	Many bones symmetrically.
Ch. periostitis, osteitis, caries, and necrosis.	Common.	Early life.	One bone ; may be several in syphilis.
Tumours.	Relatively uncommon.	All ages.	Starts in one bone.
Acromegaly.	Rare.	Twenty-five to forty.	Extremities and face.
Achondroplasia.	Very rare.	Congenital.	Whole skeleton.
Pulmonary osteo-arthropathy.	Rare.	After middle age.	Arms chiefly.
Osteitis deformans.	Very rare.	After middle age (men).	Long bones.
Mollities ossium.	Very rare.	Twenty-five to thirty-five (women).	Whole skeleton.
Leontiasis ossea.	Very rare.	After middle age.	Facial and cranial bones.

child sits up, due to laxity of the ligaments ; scoliosis may ensue later. The head is square-shaped, both the frontal and parietal eminences are prominent. The anterior fontanelle may remain open after the second year (normally it should close between fifteen months and two years of age). There may be craniotabes (thinning of the skull bones) especially of the occipital region. The body may be emaciated or plump and flabby. The chest is deformed, due to sinking in at the costo-chondral junctions, so that the sternum and cartilages stand out prominently in front, and are united to the ribs along a deep lateral groove (see Fig. 34, § 86). Another groove (Harrison's sulcus) runs transversely across the chest, just above the lower costal margin. The liver and spleen are both enlarged in advanced cases ; the costal margin is everted, and the belly is prominent. The joints are loose, permitting of hypermobility. There is always gastro-intestinal trouble, and bronchitis is frequent. There is instability of the nervous system, sometimes evidenced by convulsions, tetany, or laryngismus stridulus ; nodding spasm is more rarely present.

Diagnosis.—The disease may have to be diagnosed from hereditary syphilis, in which there may be enlargement of the epiphyses, but this occurs usually only in one bone, and is accompanied by other undoubted signs of syphilis. It may also need to be distinguished from the other diseases of this group which affect children. Infantile paralysis soon exhibits muscular wasting. Achondroplasia (§ 480) is a rare condition which has only recently been distinguished from rickets. In infantile scurvy (§ 443) the swellings affect the shaft rather than the epiphysis, and are painful. The diagnosis of rickets, hereditary syphilis, and hydro-

cephalus is given in the form of a table below. It is chiefly in regard to the form of the head that the diagnosis between hydrocephalus and rickets presents any difficulty.

Prognosis.—The disease when taken in hand before osseous changes are marked is readily amenable to treatment. If untreated it leads to deformity. If death occurs it is due to some of the common complications, notably pneumonia, bronchitis, or gastro-intestinal disorder, and wasting, or convulsions. Spinal, pelvic, and other deformities or hydrocephalus may result, and the growth is stunted. Genu valgum (knock-knee), genu varum (bow-leg), and flat-foot often occur.

Etiology.—Rickets rarely appears earlier than six months or later than the second year. Both sexes are equally affected. The disease is more frequent in cities, and during the dark months of the year. The recent work in Vienna has proved that sunlight is as important as diet for the prevention and the cure of rickets. The problem is complicated, but it would appear that rickets is a deficiency disease, due to the absence not only of the fat soluble A factor, but also to an unknown growth factor. The blood is poor in calcium and inorganic phosphorus, or these occur in unbalanced quantities, and it is probable that gastro-intestinal conditions play a part, causing defective absorption of calcium and phosphorus. Exposure to ultra-violet rays enables the body to retain or utilise these salts ; sunlight and various forms of lamps which are rich in ultra-violet rays can therefore prevent, or even cure, rickets. Dr. Leonard Hill states that recent research (1925) supports the view that the antirachitic body in cod liver oil and radiant energy in the form of ultra-violet light both act by facilitating the absorption of lime and phosphorus from the intestines into the blood stream.

TABLE XXIX.—DIFFERENTIAL DIAGNOSIS.

	Rickets.	Hereditary Syphilis.	Hydrocephalus
I. History.	Gastro-intestinal irritation, sweating about head. Improper feeding.	Snuffles and rash. Miscarriages in mother.	Congenital, or may be acquired after some meningeal inflammation, or due to tumour pressing on veins.
II. Age of patient.	Begins to show itself after six months and before the second year.	Symptoms first appear third week to the third month.	Congenital or acquired.
III. Shape of head.	Often compressed antero-posteriorly. Frontal eminences marked.	Irregular prominence on each frontal and parietal bone. Skull has been called natiform. Depressed bridge of nose	Bulges in all directions. General tendency to assume a globular form.
IV. Fontanelles.	Close late.	Appear to be depressed in the hollow between the four prominences.	Bulging, separation of the bones at the sutures.
V. Other peculiarities.	Epiphyseal enlargements, delayed dentition, etc.	Pegged and notched teeth. Scars about mouth, palate, etc.	Stunted growth, mental deficiency.

Treatment consists first in correcting the dietetic deficiency. Reduce carbohydrates and add raw meat juice, fresh milk, butter and cream, well chewed fresh fruit and green vegetables. Cod-liver oil contains the essential vitamin, and hence is by far the best medicine for rickets. Lime and phosphorus preparations used to be added. Secondly, exposure of the body to ultra-violet rays, such as are found in natural sunlight or artificial lamps. It has been found that even with faulty diet, rickets can be both prevented and cured by brief exposures to the mercury vapour light two or three times a week. Expert knowledge in its administration is required for this form of treatment. The child must not be allowed to walk, lest the bones yield and produce permanent curvature. Should the bones have already some degree of curvature, further yielding is prevented by placing the legs in restraining splints, which if carried beyond the feet will effectively prevent any attempt at walking or standing when the mother's back is turned. Rolled-up newspapers form a useful splint.

§ 479. II. Under **Chronic Osteitis** and **Periostitis** are included a number of tuberculous, syphilitic, and other conditions leading to caries, necrosis, and other anatomical changes in the bone. Osteitis and periostitis may be dealt with together, for although the disease may start in the bone or the periosteum it soon spreads to the other.

The *Symptoms* of osteitis and periostitis may have come on with acute pain, redness, and swelling; but more frequently they come on insidiously with hardening, thickening, or enlargement of the bone. These symptoms may be followed by softening (caries) or death of a portion of the bone (necrosis) with signs of abscess formation.

Causes and their differentiation : (1) *Traumatism* alone, without sepsis or toxæmia of some kind, is a rare cause of chronic periostitis or osteitis. Traumatism is recognised by its history, and by the fact that only one bone is affected. (2) The favourite seat of *tubercle* is the epiphysis, where it induces a chronic epiphysitis, especially in the neighbourhood of the hip or knee. Sometimes it gives rise to osteitis, and when this occurs in the fingers it results in a characteristic thickening of the phalanges known as tuberculous dactylitis. In any position it may go on to caries or necrosis. Tuberculous affection of the bones is recognised by (i.) the youthful age of the patient ; (ii.) a tuberculous history ; (iii.) by the characteristic intermitting pyrexia ; (iv.) signs of tubercle in the lungs and elsewhere ; (v.) by the chronicity of the process ; and (vi.) the frequent limitation to one bone. (3) *Syphilitic* affections of the bones are very common both in the acquired and the hereditary disease. (*a*) *Acquired syphilis* may take the form of a chronic diffuse or localised periostitis (nodes), or, on the other hand, a diffuse or a gummatous (localised) osteitis. It is recognised by (i.) the characteristic flying pains in the limbs ; (ii.) the nocturnal pains in the bones, which are such a frequent manifestation of syphilis ; and (iii.) other evidences of syphilis. (*b*) *Hereditary syphilis* may give rise in childhood and early life to the same lesions as the acquired disease. In infancy (in addition to the foregoing) chronic suppurative osteochondritis (chronic epiphysitis) is apt to arise and be mistaken for rickets. In this condition one or several bones may be affected, but it never presents the same symmetry as rickets. The deformities resulting from hereditary lesions (§ 434) and the physiognomy are very often characteristic—the bosses on the frontal and parietal bones (Parrot's nodes), the depressed bridge of the nose, scars about the angle of the mouth, Hutchinson's teeth, and perhaps keratitis. (4) Rheumatism and gout may give rise to chronic periosteal thickening, or periosteal nodes.

For the adequate *Treatment* of most of these different conditions, rest and surgical aid are necessary. The treatment of the tuberculosis, syphilis, rheumatism, and gout have already been dealt with.

III. **Tumours of Bones** may commence with pain, tenderness, and swelling like

chronic periostitis. The chief innocent tumours are **exostoses**, which may occur on almost any bone, and **enchondromata**, which are commonest on the metacarpals and phalanges. Both are usually multiple. The *malignant* tumours are either sarcoma (especially myeloid sarcoma) or carcinoma. In both the swelling of the bone is more rapid, and reaches a greater degree than in any of the other causes of swelling above mentioned, and as a rule they are limited, at any rate at first, to a single bone. Spontaneous fractures may occur.

§ 480. Certain rare forms of chronic bone disease must be mentioned.

IV. **Acromegaly** is a rare disease first described by Dr. Pierre Marie, leading to enlargement of the skeleton. The patients generally apply for treatment for some other malady, though sometimes they apply on account of the awkwardness of their movements, and sometimes they complain of obscure pains in the limbs. The aspect is very characteristic. The bones and other tissues of the hands and feet become markedly elongated and hypertrophied, though the growth is so gradual as to escape the patient's notice. The cranium is increased, but not so much in proportion as the face, which is egg-shaped, the lower jaw representing the large end of the egg. The lower jaw especially is enlarged, and may project beyond the upper jaw. The nasal bones are also enlarged, whilst the thickening of the soft parts causes hypertrophy of the ears, eyelids, nostrils, and tongue. Later in the disease there may be a similar enlargement of the bones of the limbs, and the thorax, and kyphosis of the spine. Sometimes there is temporal hemianopsia, with gradual optic atrophy.

Diagnosis.—Myxœdema resembles acromegaly, but it is known by its round or "moon-shaped" face, the dry skin, and the absence of all bony enlargement or muscular weakness. Pulmonary osteo-arthropathy (see below).

Acromegaly occurs rather more frequently in women, generally beginning about the twenty-fifth year. Changes in the pituitary body, either hypertrophy or tumour, have been found in all the fatal cases. Gigantism and acromegaly are both due to disordered function of the pituitary glands. In the skulls of certain giants the sella turcica has been found to be considerably enlarged. In some cases there have also been changes in the thymus or the thyroid glands, and mediastinal dulness has been made out during life in the position of an enlarged thymus.

Treatment.—Acromegaly runs a very prolonged course of many years, and no known treatment seems to affect it. Extract of thyroid may be tried. The patient generally dies of some intercurrent malady.

V. **Achondroplasia** (Synonyms : Fœtal Rickets, Chondrodystrophia Fœtalis) is a rare condition of infancy leading to dwarfism and generalised deformity, which until a few years ago was probably confused with cretinism on the one hand, or with the deformity resulting from rickets on the other. Fig. 123 represents a case exhibited at the Clinical Society of London by Mr. W. Turner, to whom the author is indebted for the photographs. There is a generalised symmetrical shortening of the diaphyses (producing characteristic shortening of the limbs) with considerable thickening of the epiphyses (producing enlargement of the articulations), due to hyperplasia of the cartilaginous ends of the bones. Consequently the stature is stunted, the fingers and toes taper and are abducted from one another, the cranium is large, the face small, and the bridge of the nose depressed. There is a characteristic waddling gait. The disease is congenital, and dates from birth. The mental deficiency, facial aspect, and the changes in the hair and skin characteristic of cretinism are absent, and cases do not exhibit the constitutional symptoms or characteristic changes of rickets in the skull. It is ascribed to a premature union of the diaphysis and epiphysis, so that lengthening of the large bones is arrested.

VI. **Pulmonary Osteo-arthropathy** is a chronic hyperplasia sometimes associated with chronic pulmonary disorders. There is enlargement of the hands and feet, and of the lower ends of the long bones of the legs and forearms, but the face and head are not enlarged. The nails are curved over the enlarged terminal phalanges, "filbert nails."

VII. **Osteitis Deformans** (Synonym : Paget's Disease) is a somewhat rare disease

coming on after middle life, mostly in males, and consisting of a very chronic enlarge-
ment of the bones, both in diameter and in length. The histological change is a rare-
fying osteitis with enlargement of the Haversian spaces. It affects the cranium
(not the face), spine, limb, bones, and clavicle. It becomes manifest to the patient
by the fact that he frequently has to change the size of his hat. Sometimes rheumatic
pains in the bones occur. The head is projected forwards, associated with kyphosis
in the dorso-cervical region, so that the attitude is characteristic. The base of the
chest is expanded, the abdomen diamond-shaped, and crossed by a deep transverse
sulcus, the hips are widened, and the legs are bowed outward and forward.
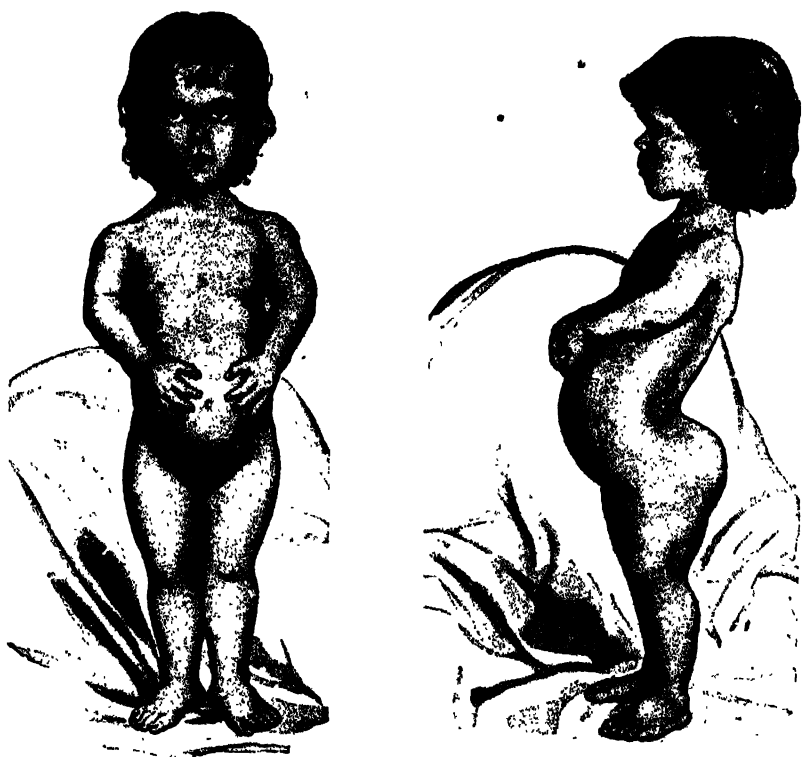
FIG. 123.—A case of ACHONDROPLASIA exhibited by Mr. W. Turner at the Clinical Society of London
(Trans. Clin. Soc. Lond., vol. xxxii., 1899).

VIII. **Mollities Ossium** (Synonym: Osteomalacia) is a progressive disorder of the
bony system, due to gradual decalcification and weakening of the skeleton, which
results in considerable deformities and contortions, owing mainly to muscular action.
It occurs in women between twenty-five and thirty-five years of age, mostly after
pregnancy. The early symptoms consist of wandering pains in the limbs and trunk,
worse at night, with weakness of the limbs. In the course of a few months there is
bending of the bones; spontaneous fractures and distortions may occur. The stature
is diminished from the involvement of the spine. Death usually occurs from respira-
tory complications owing to the fracture of the ribs. Cures have resulted in some cases
after oöphorectomy.

IX. **Leontiasis Ossea** is the term given to a rare condition in which there are

symmetrical hyperostoses of the facial bones and skull, which encroach upon the cranial cavity, and so may lead to death.

X. **Multiple Myeloma** (Synonyms : Kahler's disease, Myelopathic Albumosuria) is a disease of the bony skeleton, due to a diffuse new growth of the myeloid tissues, which erodes the ivory elements of the bones and displaces the blood-making marrow, with resulting anæmia, and tumours on the bones. In the early stage the chief symptom may be pain, not as a rule localised to any one position, but varying from day to day. The patient complains of gradually increasing debility; and the bones may bend or fracture without apparent cause. The urine is found to contain the Bence-Jones protein (see § 312, albumosuria), and may be milky when passed or on standing. Sometimes the discovery of this constituent in the urine is the means of diagnosing the disease in an early stage, when nothing beyond weakness and pains are complained of. The disease has in the past been confused with Mollities Ossium. Myelopathic albumosuria affects for the most part males over the age of thirty-five. No treatment is known. The disease is fatal in about one to four years.

XI. **Osteogenesis Imperfecta** is a condition occurring during intra-uterine life or soon after birth, manifested clinically by multiple fractures without sufficient cause, or sometimes by acute bending of the bones. The fractures may not heal, or heal only with difficulty and much callous formation.

XII. **Cleido-cranio-dysostosis** is a congenital defect. There is absence of bone at the outer third of the clavicle, with persistence of the acromio-clavicular ligament, and deficient ossification of the cranial bones.

XIII. **Brittle Bones** and **Blue Sclerotics.**—A peculiar syndrome in which unusually blue sclerotics—similar to those of congenital heart disease—are associated with brittle bones, leading to multiple fractures, has been noted by various writers. It is possible that syphilis plays some part in its etiology. The fractures heal well, and the general health is not otherwise interfered with.

XIV. **Perthes disease** (pseudo-coxalgia) is a rare disease which affects the epiphysis of the head of the femur. It occurs in children under twelve, and is evidenced first by a limp and limited abduction; later there is shortening and muscular wasting. X ray decides the diagnosis. There is usually spontaneous recovery in a few years.

CHAPTER XVIII

THE SKIN

THE skin is subject to the same diseases as other epithelial structures, but the circumstance which strikes the thinking student is that, although various skin diseases have, as it were, written their own characters on the surface of the body in full view of the observer, we have hitherto learned no more about the pathology of morbid processes which occur in the integument than we have about those which take place in other parts of the body.

Another circumstance which increases the mystery surrounding skin disease is the immense number of names adopted by dermatologists for the same or but slightly different diseases. It has not been possible to give all the synonyms for the various recognised types of disease in the following pages. This tendency to cut up a disease into an infinitude of varieties and subvarieties seems at last to be on the wane, and a more scientific tendency to trace several differently named varieties to one morbid process has set in.

PART A. SYMPTOMATOLOGY

The cardinal symptom of skin affections consists of an ERUPTION with or without SUBJECTIVE SYMPTOMS. The subjective symptoms of skin diseases are of relatively less importance for diagnostic purposes, because the morbid process itself is before us. There is, however, one subjective symptom which attends a great many skin diseases—namely, PRURITUS (itching). Anæsthesia and other disorders of sensibility will be dealt with among diseases of the nervous system.

§ 481. **Pruritus** is the Latin word for itching, and that is the sense in which it is used here.

There are three groups of *Causes* of itching:

(a) Pruritus may be *secondary* to some visible skin disease, and in that case the itching is localised to the neighbourhood of the eruption. Some eruptions are invariably attended by itching, such as urticaria, eczema and most acute conditions which progress rapidly. Other diseases are generally unattended by itching, such as syphilis, psoriasis, and most chronic conditions which evolve their course slowly.

(b) Various *local conditions* may produce more or less localised itching:

(1) *Discharges* or secretions from nasal, buccal, or anal orifices, *e.g.*, pruritus ani, pruritus scroti, pruritus pudendi. In many of these cases there is also a certain amount of localised eczema, which is possibly also the result of the discharge or sweat. (2) A *rough garment*, such as a new flannel shirt or certain dyed articles, may produce intolerable itching in delicate skins. (3) Various *parasites* give rise to pruritus. With *scabies* there is also a characteristic eruption localised chiefly to the flexures of the joints. In *phtheiriasis* (due to pediculi corporis) the eruption is generalised, though most intense across the shoulders ; the *flea*, the *harvest-bug*, *pediculus pubis*, and other parasites cause intense itching.

(c) With *idiopathic* or internal causes the itching is generalised, and may or may not be accompanied by a certain amount of generalised eruption of papules (see Prurigo, § 495). Among the causes may be mentioned gout, certain articles of food (*e.g.*, shell-fish, eggs, cheese, excess or deficiency of salt); jaundice, digestive disorders, diabetes, Hodgkin's disease, kidney disease, pregnancy, nervous irritability, constipation, and old age. Pruritus, with congestion of the nasal and intestinal mucosa, also occurs in slight degrees of anaphylaxis ; certain foods can cause it. In some cases itching is due to neurosis ; thus nervous persons complain of much itching long after the causal condition has been removed. The *Treatment* of pruritus is given under Prurigo.

PART B. PHYSICAL EXAMINATION

The APPARATUS required for the investigation of skin diseases is simple, and consists of a good lens some 3 inches in diameter, a microscope with accessories, and the means of histological examination. A pair of flat forceps is useful for removing scales, hair, or parasites. A flat glass slide may be used or the skin may be stretched to ascertain if the spots disappear on pressure.

HISTOLOGICAL EXAMINATION is of great use in many cases, and frequently enables one to diagnose a lesion with certainty ; a small piece of the diseased skin can be removed without causing appreciable pain if the part be first frozen by ethyl chloride. As the skin freezes pinch up the fold required with the fingers so that it retains its shape, then take hold of it with a forceps, and cut with curved scissors a tiny piece (including all the layers of the skin), which can be put into alcohol, then collodion or paraffin for section cutting.

§ 482. The points to investigate in any given case of skin eruption are : I. The size and appearance of the prevailing elements ; II. What it feels like, and whether it disappears under pressure ; III. The distribution and symmetry of the eruption ; IV. Subjective symptoms ; V. The duration and evolution of the eruption ; and VI. Its etiology.

I. The **Character and Size of the Prevailing Elements.**—The spots are *never all quite alike*, being modified by the age of each spot, the locality affected, and the conditions to which it has been subjected (*e.g.*, scratching or pressure). It is therefore of the highest importance *to examine every part* of the eruption. Patients may object to undress and the physician

may grudge the time, but these considerations should never be allowed to weigh. The most convenient clinical classification of skin diseases is based upon the nature of the elementary lesions, a list of which is given below.

§ 483. The principal **elementary lesions** which appear on the skin are as follows : There are three varieties of primary lesions, and three which arise secondarily to these.

1. A *macule* (or macula) is a spot of congestion not elevated above the surface of the skin ; *roseola* is a generalised eruption of macules ; *erythema* is a larger area of congestion with fading edges. A *wheal* is a spot of congestion accompanied by slight exudation beneath the skin ; it is also called *urtica*, and a generalised eruption of wheals is called *urticaria* or " nettle-rash," because it resembles nettle stings. When large, the wheal is white in the centre and red around.

2. A *papule* (or pimple) is a small solid elevation of skin, conical, round-topped, or flat. A *lenticular* papule is a large flat-topped papule. A tubercle, or *nodule*, as it may be better called, to avoid confusion with the lesion of tuberculosis, is larger than a papule, but not large enough to be called a tumour.

3. A *vesicle* is a collection of serous fluid beneath the cuticle. A *bulla* is a large vesicle. A *pustule* is a collection of purulent fluid beneath the cuticle.

The SECONDARY lesions are :

1. A *scale* or squame is the exfoliation of cuticle which occurs after a congestion or inflammation of the skin, or it may be the product of pathological processes special to the skin, such as cornification, or hyper-keratosis. In a sense a scale may be a primary lesion.

2. A *crust* or scab is dried serum or pus.

3. *Fissures*, ulcers, cracks, excoriations are breaches of the surface. *Cicatrices* or scars may result from these when a sufficient extent or depth of skin is involved.

PIGMENTARY ALTERATIONS are known as *chloasma* when there is a broad region of excessive pigment ; *leucoderma*, when there is an area of skin devoid of normal pigment ; *melanoderma*, when there is an area of increased pigmentation. *Ephelis* is a freckle. *Nævus* is a mole or birth-mark, either pigmented, hairy, or vascular. A dilatation of the superficial vessels of the skin is known as *telangiectasis*. *Petechiæ* are small spots of hæmorrhage into the skin. *Ecchymoses* are larger patches of extra-vasated blood which go through the changes of colour characteristic of a bruise. A *comedo* or " blackhead " is a little black plug of inspissated sebum blocking the orifice of a sebaceous gland.

The fundamental histological changes of the skin are congestion (hyperæmia) with or without exudation, inflammation, and infiltration. If the lesion consists of congestion, such as roseola, or urticaria, or simple *inflammation* without infiltration, such as eczema, it disappears on pressure. If, on the other hand, there be definite *infiltration* or neoplastic deposit, as in lupus and syphilis, or if there be *hæmorrhage*

into the skin, the colour does not disappear when the skin is pressed by the finger or a glass slide, or stretched. This is a point of much significance in the diagnosis of skin diseases. The secondary consequences of inflammation in the skin are, as elsewhere, three in number. If the inflammation does not undergo resolution, there may be (1) *suppuration* leading to the formation of pustules, ulcers, etc. ; (2) *necrosis*, as in the centre of boils and carbuncles ; or (3) *organisation*, as in the case of the various scars, hypertrophies, or scleroderma. In addition to the primary lesions just referred to—congestion, inflammation, and infiltration and their consequences—which occur in the skin as elsewhere, there are at least four processes special to the skin. 1. *Hyperkeratosis* is an increased deposit of kerato-hyaline material leading to an increased cornification of the surface cells of the epidermis, and a scaliness of the surface as in pityriasis and psoriasis. 2. *Parakeratosis* is the irregular or deficient cornification which occurs, for instance, in eczema. Here the prickle cells, instead of going through the regular process of cornification by the deposit of kerato-hyaline granules in their interior, and their gradual conversion into dry, horny, non-nuclear cells, remain moist and succulent (though dry on their exterior), and retain their nuclei. They adhere to one another, being moister, and are shed in masses of crusts and scales instead of being shed singly and imperceptibly. 3. *Acanthosis* is a term applied to the increased proliferation of the prickle cells by increased mitosis (karyokinesis), resulting in an increased thickness in the epithelial layers of the cuticle. 2 and 3 are found in all kinds of eczema, 2 chiefly in dry eczema ; 3 is met with in moist eczema.

II. What does the **eruption feel like,** and does it **disappear on pressure** ? Infiltrating lesions feel hard, and do not entirely disappear on pressure, as is evident from the histological characters (*vide supra*). A faint purpuric eruption may thus be diagnosed from an erythema.

III. The **distribution, position** and **symmetry of the eruption** is important for purposes of diagnosis, and it is therefore most essential to examine the whole of the eruption. Many diseases may be recognised by the position in which the elements predominate, and Figs. 124 and 125 will aid the student to remember the parts most frequently affected by certain eruptions. Some diseases are always more or less generalised—*e.g.*, urticaria and the exanthemata, and this generalised distribution usually indicates a toxaemic or idiopathic cause. Others, while sometimes affecting the whole body, have a preference for certain parts—*e.g.*, psoriasis for the knees and elbows, acne for the head and shoulders. Various words are used to describe the distribution : thus, *punctate* when the eruption is dotted about, *discrete* when the elements are separate, *confluent* when they run together, *gyrate* or crescentic when they are arranged in wavy lines or segments of circles, *circinate* or annular when they are in circles, *corymbose* when grouped into clusters.

Any **symmetry** of arrangement on the two sides of the body should be carefully observed, though its significance must not be overrated. It may indicate that some constitutional or blood change is in operation, as in the earlier eruptions of syphilis. Symmetry may also indicate that some nervous or neuro-vascular cause is in operation, as in certain erythematous eruptions. But perhaps the commonest cause of symmetry is the fact that both of the parts involved are exposed to the same extraneous conditions, as in eczema of both hands due to washing in strong solutions of soda.

IV. The presence of **Subjective Symptoms** must be inquired into, such as itching, burning, smarting, etc. Syphilitic eruptions do not usually itch, a feature which helps to distinguish them from the corresponding non-syphilitic rash. The majority of skin diseases are *unattended by constitutional symptoms*, if we except tuberculous and syphilitic eruptions, and the eruptive fevers.

V. The **Duration of the Eruption** and the history of its **Evolution** must be investigated. The rate at which a disease has developed is a most important aid to diagnosis. For instance, lupus vulgaris will not produce so extensive a lesion in the course of years as a facial syphilide which resembles it will produce in the course of weeks or months. It must be remembered also that during its progress a skin disease may alter its appearance considerably; a lesion which starts as a papule may become a vesicle and then a pustule, as in small-pox.

VI. The **Etiology** must also be inquired into. First as regards PREDISPOSING causes: 1. The *age* of the patient. Lupus vulgaris nearly always starts in early life, but lupus erythematosus rarely starts until middle life. 2. *Sex* does not aid us much in diagnosis. 3. *Heredity* is not a potent factor in skin disease, though ichthyosis, psoriasis, cancer, and albinism have been traced in families. 4. The *occupation* of a patient may result in certain skin diseases by a want of cleanliness, or may cause various forms of eczema of the hands (grocer's itch, baker's itch, etc.).

Among the EXCITING causes—1. *Traumatic conditions* frequently produce a lesion which is indistinguishable from eczema, and friction and scratching may modify the characters of an eruption very considerably. 2. *Parasites* produce eruptions which have special characters. 3. *Vegetable organisms*, fungi, on the surface of the body cause ringworm, favus, and other diseases. 4. *Bacteria* introduced *into the body* are the cause of the exanthemata. 5. *Gout* and other toxæmic conditions are the causes of some eruptions—*e.g*, urticaria, erythema, and prurigo. 6. Many *drugs* are attended by characteristic eruptions (§ 488). 7. *Diseases of the internal organs* may produce eruptions, especially digestive disturbances (urticaria), disease of the peripheral nerves and their ganglia (herpes and glossy skin, and other trophic changes), acute and chronic renal disease, diseases of the liver, and other abdominal diseases.

PART C. DIAGNOSIS, PROGNOSIS, AND TREATMENT OF SKIN DISEASES

§ **484. Routine Procedure and Classification.**—The LEADING SYMPTOM is generally before our eyes. The HISTORY, DURATION, and MODE OF EVOLUTION can be inquired into while the patient undresses. Then we proceed to the PHYSICAL EXAMINATION as described in Part B.

FRONT.   BACK.

FIGS. 124 and 125.—DIAGRAM showing the parts most frequently affected by certain eruptions.

C.M.   Y Y

GROUP I. ERUPTIONS USUALLY DRY

§ 485. Urticaria (" nettle-rash ") is a generalised eruption which consists of firm pinkish swellings, white in the centre when scratched, the typical wheals, which are of more or less evanescent character, rarely lasting more than a few hours. The rapid onset and disappearance of the individual lesions is characteristic. Patients come complaining of the *history* of such an eruption accompanied by intolerable itching. Sometimes, although there are no wheals visible, these can readily be produced by drawing a point across the skin (dermatographia or urticaria factitia). This latter condition may very frequently be found in association with the exanthems and erythemas. It is also present in states of cerebral congestion (meningitis) and constitutes the tâche cérébrale of Trousseau.

Etiology.—Urticaria may be due to external or internal causes. (1) The bite of some insects, bugs, mosquitoes, the stings of nettles or jelly-fish. In rare cases a bath is followed by urticaria. (2) Nervous causes are rare, but some persons develop urticaria on meeting a stranger or before addressing a public gathering. (3) Anaphylaxis, or susceptibility to a foreign protein, congenital or acquired. In certain people and families this may be associated with a tendency to hay fever, asthma and eczema. The commonest foods to cause urticaria in those who are sensitive to the foreign protein are eggs, shell-fish, pork, acid fruits or wines. (4) Sometimes the foreign protein is bacterial ; I have seen two cases where severe urticaria disappeared on removal of apical abscesses of the teeth. (5) Serum injections. (6) Gastro-intestinal disturbance caused by decomposed foods such as bad fish or tinned foods, or dyspepsia after a heavy or indigestible meal. (7) After enemata. (8) Drugs (§ 488).

Varieties.—(1) There is an acute, and chronic form of the affection ; the first-named consists of a transient attack lasting a few hours or days ; in the chronic or more properly the *recurrent* form (urticaria perstans) there are constantly recurring attacks. (2) Urticaria papulosa (§ 495), and (3) Urticaria pigmentosa (§ 506). (4) Urticaria bullosa is a rare variety with vesicles or bullæ, met with chiefly in children. (5) In giant urticaria or Quincke's disease the eruption is more persistent and consists of circular white œdematous spots or swellings in the skin. (6) Angio-neurotic œdema consists of still larger swellings affecting the loose subcutaneous tissue. Angioneurotic œdema may cause such large swellings that when it occurs in certain regions there may be danger to life ; this is seen especially when the mucous membranes are involved. It may occur in association with purpura, and is probably an anaphylactic phenomenon. (7) " Serum Disease "—The form appearing after serum injections is associated with a group of symptoms which indicate hypersensitiveness or anaphylaxis (§ 418). The symptoms are usually slight after a first injection. About eight or ten days after the prophylactic dose of antitetanic serum soldiers often developed a local or general erythematous

and urticarial rash, with temperature and malaise for a few days. If the injection be repeated after the incubation period of the first is over, symptoms of anaphylaxis may be pronounced, with urticaria, fever, joint and glandular swellings, and œdema ; in rare cases dyspnœa, collapse and even death occur.

Prognosis and Treatment.—The disease as usually met with subsides in a few days to a week, and a brisk saline purge is all that is necessary. Relapsing cases are difficult to cope with, and in these strict attention to the diet and the digestive organs is called for. In cases where the coagulation time of the blood is delayed, calcium chloride hastens the cure, in my experience, when given in large doses. In chronic cases the cause must be searched for and removed.

(b) *Eruptions which usually consist of Macules or Erythema*

Generalised.	Localised.
I Exanthemata	I Rosacea.
II Roseola (simplex and syphilitica)	II Lupus erythematosus.
III Erythema scarlatinoides.	III. Erythema nodosum
IV. Drug eruptions.	IV. Erythematous eczema, X ray dermatitis, Erythema paratrimma,
V Erythema multiforme.	E. faciei, E. traumaticum, E. caloricum, E. Pernio, and other varieties of E. multiforme, Macular Leprosy, and Pellagra

The early stages of eczema and of other eruptions to be mentioned hereafter may take the form of an erythema.

I. The **Exanthemata** or eruptive fevers are fully described in Chapter XV, where they form Group I of the acute specific fevers.

§ **496.** II. **Roseola** is a term employed to designate a generalised eruption consisting of patches of congestion, more or less marginated, varying in size from a pin's head to a lentil. Two varieties are described.

Roseola Simplex may resemble measles, and indeed, its chief importance is in connection with the diagnosis from this disease (*q.v*) : it gives rise to a considerable amount of itching and irritation, with usually a slight degree of constitutional and gastro-intestinal disturbance. It may occur in childhood under the same conditions as urticaria, and is therefore predisposed to by gastric disturbance. The occurrence of such an eruption when small-pox is prevalent should make one suspect the initial eruptions of that disease. It is one of the commonest rashes associated with vaccination. Drugs, such as copaiba, may cause it. *Treatment* consists in administering an aperient, salines, and diuretics, and in correcting any concurrent digestive disorder.

Roseola Syphilitica is the earliest of the syphilitic skin eruptions, occurring three to six weeks after infection. It appears upon the trunk, chiefly its anterior aspect, the chest, the flexures of the limbs and the palms and soles, as rosy or *dusky* red macules, disappearing on pressure, rounded, oval or irregular in shape with fading edges, varying in size from a pea

to a shilling. A degree of pigmentation may be left behind. Sometimes the eruption is so faint that it is overlooked. It becomes better marked after a bath or when the skin is exposed to cold. It may last from a few days to a few weeks. It is *diagnosed* by the history and other signs of syphilis. *Non-syphilitic roseola* undergoes rapid changes in size and shape; *pityriasis versicolor* can be scraped off and is fawn-coloured; *seborrhaic eczema* develops greasy scales on the surface, and forms spots of irregular size and outline.

§ 487. III. **Erythema Scarlatinoides,** as its name implies, consists of a widespread rash, resembling scarlet fever, preceded and accompanied by fever and constitutional
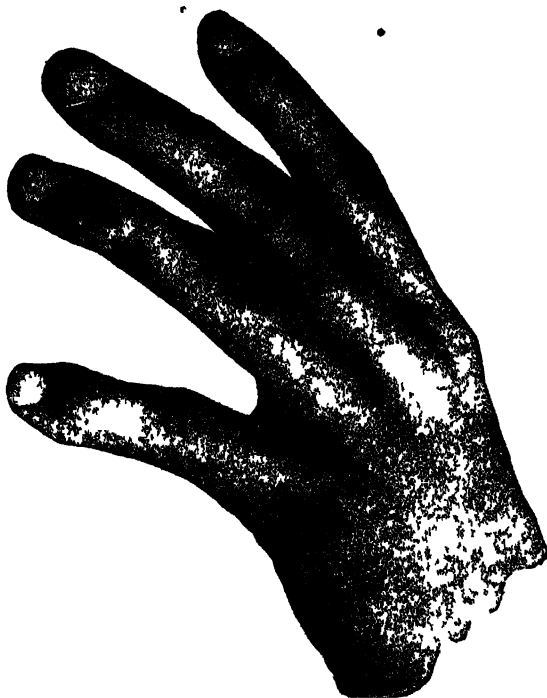
FIG 126 —ERYTHEMA IRIS on the hand of a single woman twenty-three years of age.

disturbance, and followed by desquamation. So-called "surgical scarlatina" is probably identical with this condition. The chief causes are septicæmia, intestinal disorders, enemata of soap or other substances, the ingestion of ptomaines and other toxins, certain drugs (see below), rheumatism, gonorrhœa, and sewer-gas. The *Diagnosis* from scarlet fever is difficult only in severe cases. In erythema there is less constitutional disturbance, no strawberry tongue, and there is a tendency to relapse.

§ 488. IV. **Drug Eruptions.**—An idiosyncrasy with regard to certain drugs, whether taken by mouth or applied externally, is shown by some individuals, and is manifested by the appearance of a rash, which disappears on the withdrawal of the drug. Two things will be noticed in the list below : (1) That by far the commonest eruptions

are erythema and its congeners ; and (2) that the iodides and bromides are among the commonest drugs to produce eruptions ; they may even produce a frambœsial eruption resembling gumma.　The chief eruptions produced by the internal administration of drugs are as follows :

Papulo-Pustules : Bromide and iodide of potassium (chiefly on the face), occasionally sulphide of calcium, antimony, arsenic, and mercury.

Erythema : Antipyrin, antitoxins, copaiba, cubebs, santal, turpentine, opium, chloral hydrate, belladonna, atropine, quinine, mercury, salicylic acid and sodium salicylate, boracic acid ; iodoform and carbolic acid by absorption from wound dressings.　And in the author's opinion certain toxins absorbed with milk.

Urticaria : Quinine, copaiba, turpentine, valerian, santonin, sodium salicylate, benzoic, salicylic and tannic acids.

Erysipelatoid (erythema with infiltration or œdema of the skin) : Bromide and iodide of potassium, quinine, iodoform, mercury, boracic and carbolic acids (aconite, oil of cade, chrysarobin, and carbolic acid applied externally).

Herpes : Arsenic.

Bullæ : Antipyrin, arsenic, the balsams, mercury.

Purpura : Iodide of potassium, chlorate of potash, chloral hydrate, chloroform, copaiba. •

Pigmentation : Silver nitrate, arsenic, antipyrin.

Epidermic Thickening : Arsenic and borax.

§ 489. V. Erythema Multiforme is an affection characterised by erythematous efflorescences, varying in form and size, localised usually upon the backs of the hands and forearms, dorsum of feet and legs, and sometimes on the face, neck, and the trunk, and accompanied usually by lassitude and ill-health.　The lesions vary in size from a lentil to the palm of the hand.　They belong to the *congestive* form of eruption, being slightly raised, with fading edges.　The centre is the highest part, is usually livid, and sometimes hæmorrhagic.　There is usually a feeling of burning or formication.　Many varieties have been described, such as *E.'gyrata*, when the blotches fade in the centre, coalesce with neighbouring ones, and form wavy or gyrate lines.　*E. iris* (herpes iris, herpes circinatus) is a form in which a vesicle forms in the centre, with concentric rings of purple and white and red around it (Fig. 126).　In *E. bullosum* bullæ form.　*E. nodosum* is described in § 492.　Erythema is known from urticaria by its deep red coloration, by its more localised distribution, the larger size and more permanent character of the lesions, less itching, and more marked constitutional symptoms.　Young people and males are more prone to the affection.　It is commoner in the spring and autumn.　The *course* of the disease varies, but each erythematous patch lasts eight to ten days, and they continue to appear for two to six weeks.　Each may leave temporary brown pigmentation, and desquamation may occur as they fade.

Etiology.—Sometimes a definite history is obtained of onset after food poisoning. In other cases streptococcal foci of infection are found, especially in the naso-pharynx or mouth.　In the majority of cases complications are rare.

Treatment.—Locally, cooling lotions alone are required.　Rest is indicated lest any generalised infection attack the organs.　Internally, calcium chloride, calcium lactate and quinine give in most cases excellent results.　Quinine gr. v. (0·3) is given t.i.d. ; calcium chloride gr. xx. (1·3), calcium lactate gr. x. (0 6) after meals.　The digestive system must be regulated first.　Salicylate of soda with sodium bicarbonate aid some cases, possibly by their action upon the liver.　The cause should be sought for and removed between the attacks.

Erythema of more or less LOCALISED *distribution.*

§ 490. I. Rosacea (Synonyms : Acne Rosacea, Acne Erythematosa, Gutta Rosacea Kupeferrosa) presents three stages : (1) Simple congestion or erythema attacking the nose and adjacent parts of the cheek, often

worse after meals or exposure to heat. (2) In the next stage dilated vessels (telangiectases) appear upon the surface, and after some time, inflamed sebaceous follicles (papules and pustules). (3) The third stage involves considerable hypertrophy, leading to the formation of nodules (rhinophyma) which are sometimes of great size. Rosacea runs a prolonged course ; the first stage alone may extend over many years.

The *Diagnosis* is not difficult, except in its early stage, when the erythema may be mistaken for lupus erythematosus and other kinds of erythema of the face (§ 493). The former, however, is recognised by slight scaliness ; and a lens reveals the presence of a fine " tissue-paper " scarring. The absence of comedones distinguishes it from acne vulgaris.

The *Causation* is not understood. It affects both sexes, but is more common in women. It is specially apt to affect cabmen, coachmen, mariners, and others who are exposed to the weather. Drink is a potent cause, but the disease may arise in total abstainers. Dyspepsia is a common cause, and test meals have shown in many cases a defective secretion of hydrochloric acid. In other cases it is associated with catarrhal conditions of the nose and throat, or septic infection from obscure foci.

Much of the *Treatment* of acne vulgaris (§ 494) is available for rosacea The cause, if possible, should be removed. In the first stage stomachics, alkaline carbonates, ichthyol, and dietetic rules are indicated. Several of my cases have benefited by large doses of calcium chloride ; others have responded to pituitary and thyroid. Calamine lotions and soothing remedies should be applied. Where the acid secretion is at fault large doses of hydrochloric acid are indicated. Obstinate cases arise when treatment as for boils is required. For rhinophyma scarification ameliorates even very bad cases.

§ 491. II. **Lupus Erythematosus** is the most chronic of the erythemata. The eruption has a spreading erythematous border, which as it spreads leaves a very thin permanent scar in the centre. In the first stage the disease begins with one or more small, red, slightly raised spots. By spreading at the margin and increasing in number the little patches form, in the course of many months, an irregular bluish-red area, with thin cicatricial centre and erythematous margin covered with scales, and sometimes with crusts. In another variety (seborrhœa congestiva of Hebra) there is a marginated erythema with numerous black specks, or large gaping openings of the sebaceous glands ; the central part of the skin appearing depressed, and covered with adherent dry scales, interspersed with venules. The favourite seats of the eruption are the cheeks and bridge of the nose (butterfly distribution); then other parts of the face and forehead, the lips, ears, scalp (where the scar leaves permanent bald patches), the extensor surfaces of the hands, fingers, and toes, and more rarely on other parts of the body. The patches are generally symmetrical. In rare cases the erythematous patches become rapidly widespread over the body, and severe constitutional symptoms are present.

Etiology.—The disease is more frequent in women than men, and rarely occurs under twenty, an important fact in the diagnosis from *lupus vulgaris*, which almost always appears before or during adolescence. Sunlight may excite the lesions in predisposed persons. Some say lupus erythematosus is connected with tubercle, but the bacillus has never been found in the lesions. Dr. H. Barber attributes it to a

streptococcal infection, chiefly of tonsillar origin. The *Diagnosis* from lupus vulgaris is given in tabular form (§ 521). Before cicatrices appear it may be hard to distinguish from *Rosacea*. The acute disseminated type may be confused with *Erythema Multiforme*. *Prognosis.*—L. erythematosus extends over ten or twenty years ; always terminates in cicatricial changes in the skin, and permanent baldness of a hairy part. Beyond the disfigurement the chronic form of the disease is not serious. The acute disseminate variety usually terminates fatally.

Treatment.—In the early stage we must employ soothing remedies (*vide* acute eczema). Where little infiltration, try salicylic and carbolic ointment, or salicylic collodion, or plaster mull.* Painting with carbolic or carbol-camphor, or sulphur pastes, etc., gives rise to a reactionary inflammation and swelling which runs its course in a few days, then results in considerable improvement. Linear scarification, diathermy, zinc and copper cataphoresis, and carbon dioxide snow have also given satisfactory results. Internally, quinine, salicylic acid, and intestinal disinfectants have given good results. Vaccine from the infecting focus is advisable.

§ 492. III. **Erythema Nodosum** is an eruption with an acute onset, consisting of erythematous lumps about the size of a pigeon's egg, occurring most frequently over both shins. The patches are round, oval, raised, non-marginated, painful, and tender. The centre is most deeply coloured, whence the purplish tint gradually fades away to the margins. There is usually some malaise and elevation of temperature ; sometimes pain in the joints and other rheumatic symptoms. Each nodule lasts one to two weeks, and successive crops may continue for a month or two. They never ulcerate. Patients are usually young women with a rheumatic tendency. The condition is known from other forms of erythema by the position of the lesion and the acute pain and tenderness. In periostitis the lesion is usually single. The disease usually runs a benign course to spontaneous recovery in a month or two, but may recur. The *Treatment* consists in the administration of salicylates, saline aperients, and after the acute symptoms have subsided, iron and quinine internally. Lead and opium lotion applied locally allays the pain. To avoid recurrence an infecting focus should be sought for and removed.

§ 493. IV. Certain specially named forms of erythema may be mentioned.

Erythematous Eczema.—Eczema is usually vesicular, but there is an erythematous variety which may run its course without presenting any vesicles. The surface of the skin is red, dry, and rough, with slight scaling. It frequently attacks the face, when the eyes may be almost closed, and is attended by burning and itching. For treatment, see Eczema, § 509.

Dermatitis due to dyes, hair dyes, and dyes in furs, especially those containing paraphenyldiamin, or work with toxic munition products such as trinitrotoluol, may cause a blotchy erythema which may pass on to acute vesiculation. Certain plants, *e.g.*, primula obconica and rhus toxicodendron, may be the cause of recurrent dermatitis in susceptible persons (dermatitis venenata).

X-ray Dermatitis may be acute, consequent on a single large dose, or chronic, after repeated small doses of X-rays. In the acute form there is erythema, swelling, sometimes bullæ, and sensations of burning or intense pain, according to the degree of the mischief. In mild chronic cases there is temporary loss of hair and pigmentation. If exposures are continued, telangiectases develop ; atrophy, cracking of the skin, warts, and indolent ulcers follow. Treatment is prevention by ensuring greater protection to those engaged in X-ray work by wearing lead foil or other material impervious to the rays. Sedative lotions and pastes hasten the recovery of acute dermatitis. Antiseptics should not be employed. For chronic forms it is necessary to order complete rest from exposure to the rays.

Bedsores (E. paratrimma) are due to pressure over prominent parts, such as the sacrum, trochanters, heels, or ankles of the bedridden, or to the pressure of a badly-

adjusted splint. A local patch of erythema appears, followed by abrasion of the skin. If the cause continues sloughs form. They are due to three causes : pressure or irritation from rubbing, perspiration and excretions in cases of incontinence, the lowered vitality of the sick and aged. In certain nerve diseases, especially myelitis, the sloughs form so rapidly that the condition is attributed to a trophic neurosis. Extreme fœtor attends the decomposition of the slough, and septicæmia may set in.

Treatment.—Good nursing can prevent bedsores. Three principles should be kept in mind : cleanliness, dryness, and relief of pressure. (i.) The parts should be carefully cleansed night and morning, and the draw-sheet pulled through immediately it becomes soiled. (ii.) After washing, the skin should be thoroughly dried by rubbing over a little methylated spirit or brandy, and well powdered. (iii.) Relief of pressure is obtained by a water-bed, ring pads, and by frequently turning the patient from side to side. If an ulcer or slough forms, these measures should be combined with the plentiful use of antiseptic lotions, and occasionally a charcoal poultice to relieve the pain and promote healing.

Erythema Faciei vel *E. fugax* is a flushing of the face which occurs chiefly in association with dyspepsia. It may form the first stage of rosacea. *E. traumaticum* develops on any part subject to long-continued pressure—*e.g.*, the garters and tight waist-bands. *E. læve* is the erythema found on the legs of dropsical persons. *E. caloricum* appears on the face from exposure to the sun and wind. *E. intertrigo* is found in parts which are opposed, such as the thighs and armpits, in infants, corpulent people, or those who perspire much. It may pass on to eczema. *E. pernio* (Synonyms : dermatitis congelationis, frostbite, chilblain) is a painful inflammatory condition of the skin of the fingers, toes, heels, or other portions of the feet or hands, caused by exposure to cold, and attended with itching and tenderness, sometimes by vesication, ulceration, or gangrene. In the form of the so-called chilblain it is prone to occur in those whose circulation is poor, and constitution feeble. Thus children and old people frequently suffer from this complaint during successive winters. *Treatment* consists in local applications of stimulating liniments, such as iodine and camphor, and internally, calcium lactate and thyroid or iron and arsenic.

Pellagra is a non-contagious disease which has long been endemic in parts of Italy, Spain and the Tyrol, and was supposed to be due to eating diseased maize. The disease appeared on the Russo-Roumanian front, where maize was the staple article of diet, and research carried out in the camps of prisoners of war tended to prove that it was a " deficiency disease." Great improvement resulted on the addition of the missing food factors to the diet. The disease at first appears in spring and disappears in winter ; later, it persists throughout the year. The eruption appears first on exposed parts : backs of hands and wrists, neck, and face, and is characterised by erythema, vesicular and bullous eruptions, pigmentation and atrophy. The eruption may appear first, or it may succeed symptoms of languor, dyspepsia, stomatitis and intestinal disturbance. Then follow headaches, vertigo, and nervous symptoms, sensory, motor, and mental, with progressive constitutional weakness. Depression, irritability, delusions, and hallucinations may ensue, and these patients often enter lunatic asylums. Some cases recover when they leave the neighbourhood at an early stage of the disease ; as a rule, the disease ends fatally in five to fifteen years.

Treatment consists in the addition of meat and lemon juice to the diet.

Macular Leprosy appears as brownish or mahogany-red patches of erythema of various sizes (§ 522).

(c.) *Eruptions which usually consist of Papular Elements*

Common.	Rarer.
I. Acne vulgaris and other forms of acne.	VI. Lichen planus.
	VII. Keratosis pilaris and follicularis.
II. Prurigo.	VIII. Milium.
III. Scabies.	IX. Lichen scrofulosorum.
IV. Papular syphilide.	X. Certain other tuberculides.

Common—(contd.)	Rarer—(contd.)
V. Skin diseases, sometimes papular at one stage :	XI. Adenoma sebaceum.
(i.) Papular eczema.	XII. Granuloma annulare.
(ii) Psoriasis and other scaly eruptions.	
(iii) Exanthemata.	
(iv.) Pustular and vesicular diseases.	
(v.) Erythematous eruptions.	
(vi) Nodular eruptions.	

§ 494. I. **Acne Vulgaris** is an eruption consisting of " blackheads " or comedones and indurated papules which may go on to suppuration, confined to the face, shoulders, and back, and sometimes the chest. Acne papules are pale red to crimson, and hard (A. indurata), varying in size from a pin's head to a small pea. These are invariably accompanied by a number of black points (comedones), which are black plugs of sebum. The papules come out one after another, and are remarkably persistent ; some go on to pustulation, or the formation of small abscesses, leaving scars if untreated. The skin of the face in individuals affected with acne is usually greasy, coarse, and dusky-looking.

Causes.—The disease in most cases starts soon after puberty, and fresh papules may continue to appear till about thirty, occasionally to middle life. Inspissated sebum blocking the follicles is the histological cause of the papules, and the change which the hair and sebaceous follicles undergo at puberty appears to be one of the factors in the causation of the disease. Sabouraud and other observers found a bacillus, which they considered causative. Indigestion and constipation aggravate the condition. In the majority of such cases there is undue acidity of the urine.

Varieties.—(1) *Acne punctata, A. indurata,* and *A. pustulosa* are stages in the diseased process, not varieties in the true sense of the word. (2) Associated with pityriasis of the scalp a crop of papules often appears on the face, almost identical with acne spots, excepting that they are smaller, softer, and not totally accompanied by comedones. Its favourite positions seem to be the chin, the furrows below the angles of the mouth, and sometimes between the scapulæ. (3) *Bromide* and *iodide acne* are indistinguishable from each other, and the individual spots resemble acne vulgaris very closely. Comedones, however, are absent, and there is a greater frequency of distribution over the chest and back, though the face is always first affected. (4) *Acne Rosacea* is mainly an erythema, at any rate in its earlier stages ; and there are no central comedones (§ 490).

5. *Acne varioliformis* is a severe variety of A. pustulosa. It occurs chiefly on the brow, and is apt to leave deep scars. 6. *A. cachecticorum* (Hebra), or *A. scrofulosorum* (Colcott Fox), or *A. necrogenica* is another pustular variety affecting chiefly the back and the extensor surfaces of the limbs, and to a less degree the face, in debilitated or scrofulous children. It is now regarded as a tuberculide. It differs markedly from A. vulgaris in its distribution, and though it may last for many years,

is generally amenable to tonic treatment and cod-liver oil It is liable to be mistaken for a syphilide, but the latter is less indolent, and there is a firmer infiltration.

The *Diagnosis* of acne from the other eruptions in this group is not usually difficult on account of (1) its characteristic position, and (2) the presence of comedones. Papular, pustular, and tubercular syphilides affecting the face are usually copper-coloured, and grouped in a serpiginous manner. Lupus vulgaris generally occupies one side of the face and presents no comedones. •

Treatment.—Indigestion and constipation aggravate acne, and must be corrected. Thyroid, calcium lactate or pituitary are indicated in certain cases. If there be much irritation soothing applications are best applied at first—e g., calamine lotion. Of all remedial agents sulphur is. the most efficacious. A sulphur ointment (20 to 40 grains (1 3–2 6) to the ounce (32)) should be rubbed on night and morning, or a sulphur lotion, or resorcin in strengths varying according to the skin of the individual. In some cases strong exfoliating remedies are best; they are applied at night and the patient remains indoors during the peeling. For pustular acne mercury is indicated, as in all suppurating affections —e.g., an ointment of 10 to 30 grains (0·6–2) to the ounce (32) To prevent scarring an antiseptic lotion should be used; each pustule must be lanced, and its interior wiped out with a pointed stick or match dipped in pure carbolic or camphor phenique. Staphylococcal vaccine injections are of value when crops of pustules resist ordinary treatment. Acne bacillus vaccine suits other cases, used alone or in combination with the staphylococcal vaccine. Washing frequently with warm water and soap, rinsing off the soap, and rubbing with a rough towel is a valuable domestic remedy. Ultra-violet light and high-frequency currents are very useful, and X-rays may be, very cautiously, tried. The fact that X-ray may lead to atrophic changes appearing many years after the exposure should decide the physician to use it only as a last resort.

§ 495. II **Prurigo** is a disease in which the leading and sometimes the only symptom is generalised itching (pruritus), but it is frequently accompanied by an eruption of papules, urticarial patches, and scratch-marks. The papules of *idiopathic prurigo* are hard, shotty, acuminate, pale red, frequently better felt than seen (giving the sensation of a nutmeg-grater), come out in crops on the extensor surfaces of the thighs and arms, the trunk, especially the back and buttocks, and only occasionally the face. Each crop lasts a week or two, and is sometimes accompanied by urticarial blotches; dermatographia can generally be elicited. The intense itching leads to scratch-marks. In the course of time prurigo is followed by a dry, rough, thickened, pigmented skin. The *Prognosis* is unfavourable, especially in the very poor, the disease recurring for years, and sometimes lasting for life.

The *Diagnosis* is simple in well-marked cases by the intensity of the itching and the condition of the skin. The prolonged course of the disease is distinctive. The eruption of prurigo very closely resembles that of

scabies (§ 496) and pediculi corporis (Fig. 127), but in scabies it is almost confined to the *flexures of the joints* instead of the extensor surfaces; in pediculosis to the *back* and *shoulders*, where it is attended by typical staining. The diagnosis from papular eczema is not always easy; eczema, however, generally prefers the flexures and flexor surfaces, while prurigo predominates on if it is not confined to the extensor aspects.

Varieties.—(1) **Prurigo infantalis** (Synonyms : Urticaria Papulosa, Lichen Infantalis, Lichen Urticatus, Prurigo Mitis) should, in the author's opinion, be classified as a variety of prurigo. The papules are small, chiefly on the back, and the urticarial element moderate. It starts about the fourth month of life, and recurs until about the fourth year. (2) In **P adolescentium** the papules are larger, the urticaria prominent, the skin brown and thickened. (3) In **P. senilis** (Synonym:
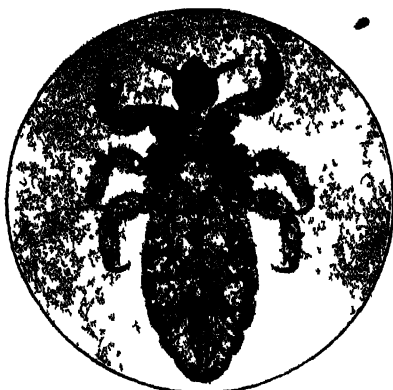

Fig 127.—PEDICULUS CORPORIS, magnified about ten times

Pruritus Senilis) the eruption may be insignificant or absent, and the irritation intractable, with a tendency to induration and purpuric complications. (4) In " *pruriginous* " *eczema*, an eczema complicates a prurigo ; (5) In *P. agrius*, the prurigo of Hebra, all the lesions are on a larger scale, the inguinal glands involved, and the general health deteriorated (6) *P. hiemalis* occurs in cold countries or in the winter only.
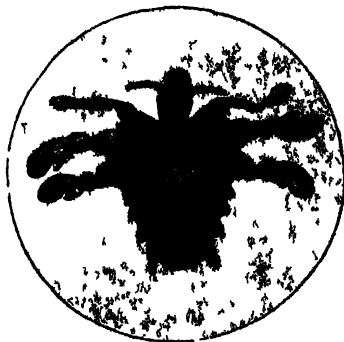
Etiology.– Prurigo is especially apt to occur at the two extremes of life.[1] Irritation of the alimentary tract is liable to start outbursts of the disease, which is greatly influenced by improper food, and unhealthy surroundings. It may be idiopathic, or secondary to scabies or pediculi. The idiopathic causes of prurigo are the same as those of pruritus (§ 481).

Fig 128.—PEDICULUS PUBIS, magnified about ten times.

Treatment.—The first indication is to discover the cause, for without carefully sifting this question no treatment can be successfully carried out. One must first exclude phtheiriasis, scabies, or other parasites.

[1] " The Pathology of Itching, and its Treatment by Large Doses of Calcium Chloride," the *Lancet*, August 1, 1896, p. 300.

Then determine whether there is any other source of local irritation, such as an unbleached cotton or flannel garment, or an acrid discharge; and finally turn to the internal or toxæmic causes. Warm weak tar baths should be taken night and morning, followed by the inunction of a mild tar or mercury ointment, to get the skin into a healthier condition, and the softest of under-garments should be worn. For local itching, carbolic acid 1 in 80, alkaline lotions, vinegar, chloral-camphor (3i. (4 gm.) of each liquefied and added to 3i. (4 gm.) starch•powder) are all useful. The diet should be plain; sugar, alcohol, and excess of meat must be avoided. Any digestive errors should be corrected, and the possibility of mucous (catarrhal) colitis borne in mind. Calcium chloride in large doses has been very efficacious in my experience. Other remedies which are worth a trial are—cannabis indica, antipyrin, valerian, belladonna, bromides (especially in nervous subjects), pilocarpin, liq. am. acet., atropine, carbolic acid, salol, salicylic acid, and other intestinal antiseptics.
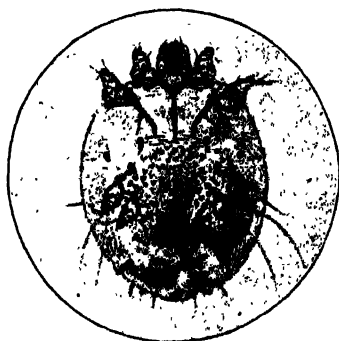

FIG. 129.—ACARUS SCABIEI (female), magnified about twenty times. The female burrows under the skin to lay her eggs, the burrows thus formed being quite typical and black with the fæces of the insect; most frequently situated on the wrist. The male roams about over the body and clothes, and is rarely captured.

§ 496. III. **Scabies** is the eruption produced by the acarus scabiei. It consists of papules and vesicles of varying sizes; the latter may go on to the formation of pustules. In addition to its multiform character, it is readily diagnosed in its typical form by (1) short white or black burrows; (2) excessive itching, and scratch-marks; (3) it always commences and predominates where the skin is thinnest—*i.e.*, between the fingers or toes, the flexures of the wrists and elbows, the breasts and lower abdomen, the axillæ, the penis, the inner side of the feet and thighs; (4) a history of contagion; and (5) the discovery of the animal or its eggs (Fig. 129). Prurigo is likely to be mistaken for scabies, but prurigo predominates on the extensor surface and outer side of the limbs. Eczema is perhaps the disease with which, in the adult, it is most frequently confused. Many experienced physicians have overlooked the parasitic origin of eczema thus produced, and it is a good rule to suspect scabies in inveterate cases of eczema. Scabies untreated may go on for an indefinite time, but it readily yields to treatment. Sulphur ointment should be thoroughly applied after a warm bath with plenty of soap, for at least four successive nights, and the underlinen and sheets should be boiled. All the clothes must be disinfected. Balsam of Peru, either pure or mixed with equal parts of vaseline, is a more expensive but equally efficacious remedy. If the sulphur produce a secondary eruption, mix it with equal parts of zinc ointment.

§ **497. IV. Papular Syphilide.**—Syphilitic eruptions are often multiform, but papules generally form the most prominent feature of all syphilitic rashes, especially in the secondary stage. The papule, indeed, forms the prototype of all syphilitic eruptions. These papules are firm, glistening, and project above the surface of the skin with a hard, infiltrated margin, and *vary in size* from a pin's head to a bean. They are of a brownish-red colour (like copper or raw ham) which does not entirely disappear on pressure. The wide variability in the size of the papules is a feature distinguishing this from other papular diseases. As they increase in size the centre often becomes depressed, or cupped. The distribution is more or less generalised, often symmetrical, but the favourite sites are the forehead, around the mouth, the *flexor aspects* of the arms, and the trunk. When near the corners of the mouth or the anus their surface may be moist, and the *secretion is highly infectious*, no matter how long after the contraction of the primary malady. Itching is rare. Other constitutional signs of syphilis may be present. The presence of shotty glands in the groin and neck and elsewhere is a valuable aid in the diagnosis of all syphilitic eruptions. They are present even when no other signs are present, and may last throughout the patient's life. Two *varieties* of papular syphilide are described according to the prevailing size of the elements, *papular syphilide* if the spots are small and numerous, *lenticular syphilide* if they are large and scanty. The former is met with more in the early, the latter in the later stages of the diseases. Large, moist, flat papules, usually seen near the anus, are called *condylomata*. Rarer forms are the *corymbose syphilide*, in which there are clusters of very small papules surrounding a central larger papule ; and small papules resembling lichen pilaris, *follicular syphilide*. The latter is very obstinate ; mercurial vapour baths may be necessary (see also §§ 434 and 520).

V. Skin Diseases sometimes Papular.—ECZEMA PAPULOSUM (Papular Eczema, Pruriginous Eczema) is a term which should be confined to papules which rapidly pass on to vesiculation, or which are associated with definite patches of eczema. Papules frequently form a stage, generally an early stage, in PSORIASIS, SEBORRHŒA, PITYRIASIS RUBRA PILARIS, SYCOSIS, in the EXANTHEMATA and ERYTHEMATA, and in XANTHOMA and URTICARIA PIGMENTOSA. These are dealt with in their respective places.

Rarer Papular Diseases

§ **498. VI. Lichen Planus** (Synonym : Lichen Ruber Planus) is an eruption consisting of flattened, angular, shiny, dull red papules, often presenting a central depression, and a greyish striation on the surface. These tend to coalesce and form irregular patches of a peculiar purplish hue. Occasionally rings are formed (*lichen annularis*). There is no exudation. When the papules disappear much pigmentation may be left behind. The eruption is frequently symmetrical, and by far the most characteristic positions are the flexor aspect of the wrists and forearms, and the inner side of the knee. Sometimes the distribution becomes rapidly widespread. The mucous membrane of the mouth is often affected and may be so for long before any lesion appears on the skin. Itching as a rule is present, and may be troublesome. By the fusion of several papules large plaques may be formed, and when these take on a raised

growth, as about the ankles, the condition, which is very intractable, is *l. hypertrophicus*,
and when warty, *l. verrucosus*. Lichen occurs mostly about middle age, and has been
noticed to appear when there is a degree of nervous debility. In Vienna two-thirds of
the patients attacked are males (Kaposi), aged ten to forty ; whereas in England the
majority of cases occur in women. There should be no difficulty in *diagnosing* lichen
planus from a *papular syphilide* or an *eczema*, on account of its typical position, angular
shape, purple colour, and flat waxy surface. The *Prognosis* as to the cure of the con-
dition is good under treatment, although this may have to be extended over many
months. The eruption remains localised to definite regions for years, but may be-
come generalised. The *Treatment* consists in the administration of arsenic and tonics
internally, and local soothing applications, such as F. 90 or 93 (see also § 530). Some
recommend small doses of hydrarg. perchlor. X-rays sometimes aid.

§ 499. VII. **Keratosis Pilaris** (Synonyms: Pityriasis Pilaris, Lichen Pilaris or
Spinosum) is an affection of the skin, generally of young adults, in which the orifices
of the hair follicles of the thicker portions of the skin—*i.e.*, on the extensor and outer
surfaces of the limbs—are occluded with corneous plugs. Hard friction, with almost
any ointment, at night, and a rough towel in the morning, will generally remedy the
condition in a few weeks.

Keratosis Follicularis (Synonyms : Darier's Disease, Psorospermosis) is a very
rare disease, due to overgrowth and degeneration of cells in the mouths of the pilo-
sebaceous follicles. The papules are at first pin-head size, resembling keratosis pilaris.
They contain in the centre a horny plug, which is difficult to remove. Some become
enlarged and hyperæmic ; others become confluent, presenting a papillomatous
surface covered by hard yellowish crusts. These may ulcerate, and the area may be
covered with a mucopurulent discharge. The disease affects first the face and head,
and after the gradual development of years appears over the sternum, spine, loins,
hypogastric and inguinal regions, and the extremities with symmetrical disposition.
The *Diagnosis* may be difficult, in the early stages, from keratosis pilaris and ichthyosis,
and in the later stages from acanthosis nigricans. *Treatment* consists in the use of
salicylic, sulphur, or other keratolytic applications, but the disease is resistant.

§ 500. Certain rare conditions—Milium, Lichen scrofulosorum, some
other tuberculides, and Adenoma sebaceum—come under the papular
rashes.

VIII. **Milium.**—The term milium is applied to an eruption of small whitish or
yellowish pearly granules about the size of a pin-point, which affect chiefly the delicate
skin under the eyes, the eyelids, cheeks, temples, scrotum, and labia. It is due to
retention within the sebaceous follicles of cholesterin and fat. *Treatment* consists
in making a small incision over the granule, and squeezing out the contents. Or
electrolysis may be employed.

IX. **Lichen Scrofulosorum** is an eruption of minute yellowish-red papules, isolated
or grouped, and occurring usually on the breast, abdomen, or back of tuberculous
subjects. They are painless, and rarely itch. It is not common after the age of
twenty. It may last, hardly noticed by the patient, for months or years, and then
subside without leaving a trace behind it. It is one of the tuberculides.

X. **Certain other rare tuberculides. Acne agminata** appears chiefly on the face, as
indolent dark red papules which involute with or without pustulation and leave a
scar. It is akin to folliclis, which occurs on the body and upper extremities, and to
other obscure pustular and scarring tuberculides. Tuberculide is the term given
to denote the eruptions which occur in persons with some manifest or latent tuber-
culous focus. It is supposed that the lesions are evidence of hypersensitisation.
They are more or less symmetrical, tend to slow but spontaneous cure, and the
patient yields a von Pirquet cuti-reaction.

XI. **Adenoma Sebaceum** is a rare disease, consisting of numerous small hemispheri-
cal elevations, discrete, grouped usually about the middle of the face. In size they

vary from a pin head to a split pea   Their surface is crimson or pinkish yellow, and associated frequently with telangiectases. They have no visible orifice. Some disappear spontaneously, leaving a small scar. The disease is almost always congenital, though it may not be observed till puberty, when it takes on fresh activity. It is said to be associated with intellectual inferiority, but this is not always the case It is due to overgrowth of the sebaceous glands and vessels The knife, electro cautery or electrolysis are necessary to destroy the growths where spontaneous involution does not occur

XII **Granuloma Annulare** is occasionally seen in young people and children. Flattened papules, white or pink, appear in ring-shaped patches, varying in size from a shilling to half a crown The centre of the patch is depressed. The lesions last long, but may disappear spontaneously or with salicylic acid ointment.

(d.) *Eruptions usually Scaly or Scurfy*

Common.

I Psoriasis
II Seborrhœic dermatitis
III Tinea circinata
IV Scaly syphilide
V Skin diseases sometimes scaly at one stage—e g , eczema, lichen, erythematous diseases

Rarer

VI. Exfoliative dermatitis
VII. Pityriasis rosea
VIII Pityriasis rubra pilaris.
IX Ichthyosis
X Erythrasma
XI Parapsoriasis

§ 501. I **Psoriasis** is a common disease, occurring as irregular patches, slightly raised, covered with copious silvery scales, unattended by any exudation, and situated chiefly on the elbows and knees. The lesion starts as a tiny papule (P. punctata) which from the first has on the top a scale, which, however, may not be visible till scratched The papule gradually enlarges (P. guttata). In a short time it reaches the size of a coin (P. nummularis) The disease generally then remains stationary for some weeks or months, and may tend to undergo spontaneous involution. The healing process usually starts at or near the centre, and gives to the eruption a circular or serpiginous appearance (P. circinata, P. gyrata). The lesion is scaly and elevated from the first, and always dry, three features which at once distinguish it from eczema, and if the top scale is scratched off bleeding hyperæmic papillæ are exposed. The distribution is extremely characteristic, being found always on the knees and elbows, frequently on the scalp, trunk, and other parts of the limbs, especially the extensor aspects, and only very rarely on the face, palms, or soles. There is little or no itching or subjective symptoms. Psoriasis of the nails causes pitting, ridging, and elevation of the free border.

Causes.—The disease is most frequent in early life, though rare under seven. Both sexes are equally affected. There is hereditary predisposition in some families. The seasonal influence varies, but those who are subject to the malady often complain that it recurs each winter or spring. The belief is gaining that the disease is a manifestation of some chronic infection.

Diagnosis.—It is important to distinguish psoriasis from *scaly syphilide*, and in many cases the character of the lesion affords no true guide. A syphilide has more infiltration; rarely affects the elbows and knees, and

generally prefers the flexor aspects, and palms, and soles ; the centre of the patches are usually depressed, stained, and healing ; the scales are scantier, less silvery, and on being scraped off, do not leave bleeding-points. In " *Seborrhœic dermatitis* " the patches are less crimson, the scales 'smaller, scantier, greasier, and more orange coloured ; they occur by preference on the shoulders and upper parts of the trunk, and if affecting the limbs are usually on the flexor aspects. The scalp may be affected in both diseases.

Prognosis.—Psoriasis is apt to disappear and to recur spontaneously at certain seasons for many years. In severe cases the eruption may spread over the whole body and cause an exfoliative dermatitis.

Treatment.—Frequent bathing, followed by removal of the scales, is an essential part of the treatment. Chrysarobin gr. xv.–ʒi. in ʒi. (1–4 in 32) is the most valuable remedy, but the objection to its use is that it stains the linen and the skin, and frequently sets up a scarlatiniform dermatitis. It must never be applied to the face or scalp. For intractable cases chrysarobin paint—20 per cent. in chloroform—may be painted on once a week, and covered with a layer of collodion. Other stimulating applications, such as tar, carbolic acid, and salicylic acid, are often quite as efficacious. Plaster mulls of the same ingredients, or of mercury, are useful. Removal of scales from the scalp is difficult, but must be enforced and followed with an ointment containing mercury. In protracted cases the patient should be detained in bed during treatment. X-rays often clear up a few obstinate patches, but should not be used for widespread disease. Internal medication is important. Thyroid extract does good where other evidences of athyroidism exist. Arsenic, cod-liver oil, salicylates, and copaiba do good in individual cases. A cause must be sought for carefully, but often eludes search. Salicylates help acute spreading cases. Arsenic is given only in chronic cases. The Danysz system of vaccine treatment succeeds with some cases, but entirely fails with others.

§ 502. II. **Seborrhœic Dermatitis** (Synonyms : Pityriasis Circinata) occurs in irregular ovoid patches, greasy, covered with brownish-red scales ; quite superficial, sometimes slightly raised, with sloping margins. They sometimes heal in the centre, forming a ringed eruption which may be mistaken for ringworm. The patches vary in size from a small pea to a crown piece, or larger. Single tiny papules may be present, brick-coloured, and soft, especially on the face. The disease is found *chiefly on the scalp*, whence it may spread on to the forehead and face, then on the sternum and back of the neck and shoulders. It is often limited to the upper part of the body, but the lower parts of the body may be affected, and the legs occasionally present typical patches. The affection is more frequent than is generally thought, being often diagnosed as ordinary eczema when from staphylococcal infection (or sensitisation) it passes on to an eczema, with vesicles and crusts. The disease is more frequent in, though not confined to, young people. It is undoubtedly contagious in

the author's experience, and is particularly apt to spread to those who occupy the same bed and whose pillows are apt to be exchanged. Professor Unna ascribed it originally to the bottle bacillus, Dr Sabouraud to the staphylococcus griseus acting in conjunction with the .bottle bacillus.

Diagnosis.— The scales of psoriasis are more silvery, and on removing them hyperæmic bleeding papillæ are seen; in seborrhœic dermatitis minute points of oozing serum may be seen on removing the moist scales.   •

The *Treatment* must not only be directed to the cure of the patches which are on the body, but to the scalp, for until this is cured the eruption will constantly recur upon the body. Frequent washing of the parts and of the clothes is necessary. A pomade of hyd ox. rub. (gr. 7 to 10 (0·4–0·6) to the ounce (32)) should be rubbed in twice a week, or a lotion of hyd perchlor. (gr. 1 (0·06) to the ounce (32)). Sulphur and oil of cade are equally efficacious (see also § 529).

Epidemic Peri-oral Eczema.—In 1895 the author had the opportunity of investigating an eruption which occurred on the faces of a large number of children in one school in the East End of London. The school contained nearly a thousand children, and quite half of these were affected in succession during three or four months The patches occurred mostly around the mouth, on the face and neck, and rarely elsewhere They were superficial, reddish, dry, scurfy, ovoid, somewhat resembling a superficial seborrhœic dermatitis. Ringworm was positively excluded. The disease readily yielded to a mild tar and mercury ointment. It certainly spread from child to child, but only one of the teachers was slightly affected (*British Medical Journal,* 1896, vol 1)

III **Tinea Circinata** may appear as small red patches, of an oval or ringed shape, slightly scaly. When the head is affected with the small-spored ringworm, these patches may often be seen on brow, neck, and shoulders. The large-spored fungus usually causes a ringed scaly eruption. Another variety, due to the epidermophyton inguinale, forms scaly patches and rings on the thighs and groins. The diagnosis is made by finding the fungus in the scales (see § 511). This form used to be described as eczema marginatum.

§ **508.** IV. **Squamous Syphilide** (Syphilitic Psoriasis).—The squamous syphilide occurs as a later stage of the papular or the tubercular syphilitic eruptions (*q.v.*), and does not constitute a separate form of eruption of itself. It is recognised by the fact that the scales are thin, scanty, and greyish, lying upon patches of stained and infiltrated skin (*i e* , the syphilitic papules) which are deep brown or copper coloured, usually round, or in the shape of *segments of circles,* having raised serpiginous scaly borders. A squamous syphilide may occur on any part of the body, but the flexor aspects and the palms or soles are particularly characteristic situations, the converse of psoriasis vulgaris. A scaly syphilide of the palms is diagnosed from dry eczema by its raised serpiginous border, with sometimes an area of normal, atrophied, or pigmented skin in its centre.

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V. **Certain skin diseases are scaly** at one stage. A *scaly* or scurfy condition of the skin, especially of the face, is produced by hard water and exposure, and in certain states of ill-health. It is also met with after scarlatina, measles, and some of the other *eruptive fevers.* In *eczema,* which is a somewhat protean disease, scales and crusts form, but the presence of exudation is its essential and differentiating quality. *Pityriasis capitis* is a scaly or scurfy condition of the scalp, sometimes called also seborrhœa sicca, or dandruff (see Diseases of the Scalp). In several varieties of *lichen,* a thin silvery scale is constantly found, although they belong really to the papular eruptions. This occurs also in papular syphilide, and it is difficult sometimes to draw the line between a papular and a scaly syphilide. *Lupus erythematosus* is attended by adherent scales and crusts.

§ 504. VI. **Exfoliative Dermatitis.**—Much discussion ranges around the use of this term and the term Pityriasis Rubra, which has been employed by some as a synonym. It is best to regard the term Exfoliative Dermatitis as implying any chronic or subacute generalised inflammatory disease of the skin, whether primary or supervening upon other cutaneous disturbance of long standing, which is characterised by hyperæmia of the entire surface, and *abundant and repeated exfoliation* of the cuticle, accompanied usually by shedding of the hair and nails. There is usually some constitutional disturbance, and the itching may be severe.

Etiology.—Occasionally, as a *secondary* affection, it may follow psoriasis, eczema, pityriasis rubra pilaris, pemphigus foliaceus, and seborrhœic dermatitis. As a *primary* condition the disease is of considerable gravity. It starts in several ways; a rapidly spreading hyperæmia of the integument is common to all. My belief is that the different varieties which have been described as separate diseases only differ in their mode of onset and etiology. *Treatment.*—Rest in bed with general treatment, as a rule with stimulants, is indicated. Ox-gall and urotropine cured one of my cases which had resisted every other form of treatment (A. §). Externally soothing baths and ointments should be used.

An **epidemic exfoliative dermatitis** which the author first had the opportunity of observing in 1891,[1] illustrated very well the wide varieties both in the severity and other features of this malady. One hundred and sixty-three cases occurred among the patients in the Paddington Infirmary, with a case mortality of 12·5 per cent. That the disease was epidemic and contagious was certain, that it was microbic seemed probable, though the author's observations on epidemics at other workhouses and infirmaries tend to show that the milk consumed by the patients was in some way the means of propagating the disease amongst them, possibly having undergone some toxic or fermentative change. **Ritter's disease,** or dermatitis exfoliativa neonatorum, is due to a streptococcal infection below the horny layer. It is usually fatal.

§ 505. VII. **Pityriasis Rosea** consists of numerous pink patches, slightly raised and pea-sized, and oval-shaped rings, with slight scaling on the pink margins, and a fawn-coloured centre. A " herald patch " usually appears on the trunk some days or even weeks before the generalised eruption, which comes out in successive crops, starting usually on the sides of the trunk, and spreading to the neck, upper arms, and thighs. It is rare on the face. Itching may be absent or severe. Pityriasis rosea runs a course of a few weeks to a few months, and disappears spontaneously. The disease occurs in both sexes, and at any age, but is most frequent in young adults. It is believed to be of parasitic origin. *Seborrhœic eczema* has greasy scales and different sites. *Tinea circinata* occurs in fewer patches, and the fungus can be found. *Psoriasis* has more infiltration and diffuse scaling. *Syphilitic roseola* has a darker colour, and is infiltrated.

Treatment.—Mild ointments of sulphur or salicylic and baths with a small amount of Condy's fluid do good. Soothing lotions are useful if itching is present.

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[1] " Monograph on Epidemic Skin Diseases," H. K. Lewis, London, 1892; Med. Soc. Trans., 1891; *Brit. Med. Journ.,* December, 1891.

§ 506. VIII. **Pityriasis Rubra Pilaris** (Devergie), Lichen Acuminatus or Lichen Ruber (Hebra), is a somewhat rare disease in which the eruption commences as tiny hard papules of hyperkeratosis involving the hair follicles, which become fused together into one reddened surface, and shed a succession of flaky scales. The distribution is fairly characteristic, as it starts where the lanugo hairs are mostly found—namely, the backs of the hands and the forearms. In this way it often presents a glove-like distribution on the upper and lower extremities, which is very characteristic. It may spread over the whole body. The progressive margin is always marked by the same tiny scale-capped papules. The disease has to be *diagnosed* from psoriasis on the one hand, and dermatitis exfoliativa on the other. It is differentiated from *psoriasis* by its distribution and by the presence of the little papules at the margin of the eruption, but is indistinguishable from dermatitis exfoliativa when the whole body is involved, except by the large flakes of epidermis in the latter. In the earlier stages dermatitis exfoliativa does not, present the small acuminate papules which *constitute the elementary lesion of P. rubra pilaris.*

The *Causes* are obscure ; the disease usually occurs before the age of twenty-one. The malady may occur in varying degrees of acuteness. Some cases are ushered in with a certain degree of constitutional disturbance, vomiting, and some pyrexia, and after lasting a few months will tend spontaneously to subside. In mild cases there are no constitutional symptoms, and the disease runs a prolonged course of many months. *Treatment.*—When it occurs in the chronic form arsenic is a remedy of some value. The same rules guide us as in the treatment of psoriasis.

§ 507. IX. **Ichthyosis** (Synonym : **Xeroderma**) may be defined as a congenital condition of the skin, characterised by an undue dryness and scaliness of the epidermis, and in some cases by the formation of wart-like outgrowths. Though congenital, the condition may not be identified till the child is some years old.

There are three *clinical types* or degrees of the affection. In the first or mild type (*Xeroderma*) there is simply an undue harshness or roughness of the skin, and consequently through life a great tendency to the supervention of "chaps," eczema, and other skin affections. It occurs chiefly on the extensor aspects. In a second type (*I. vera*) the superficial layers of the epidermis are thickened, and appear stretched ; the hardened cuticle presents fissures and cracks which, bounding polygonal areas, give to the patient the appearance of a fish or crocodile skin. The everted eyelids and nostrils, the atrophied hair and nails, and the hardened, scale-like condition of the skin are characteristic. In the third variety (*I. hystrix, I. sebacea, Papilloma lineare*), the skin presents a thickset aggregation, of little horny, wart-like processes which entangle the dirt, and present a brownish-black coloration. These are arranged in streaks, which were believed to follow the course of certain nerves, but a closer observation shows that this is not so, the disease being a developmental one. The *Diagnosis* is not difficult owing to the congenital nature of the malady. Apart from the inconvenience and the liability to eczema, the first type is not serious. In the second type the disease progresses to the age of puberty, and then remains stationary. The third type rarely shortens life, but is a disfiguring malady. No known remedy influences this disease. Resorcin 2 per cent. in glycerin amyli, vaseline, lanolin, baths, and various ointments may soften the skin, and remove the superficial scales to some extent. Thyroid seems to control milder cases to some extent.

§ 508. X. **Erythrasma** consists of defined scaly discs of a pale red, yellow, or dark brown colour. The scales can be scraped off, and are found to contain a fungus, the microsporon minutissimum. The patches are extremely chronic, and are found on the opposed surfaces of the scrotum, thighs, axillae, and mammae. They itch when perspiration is excessive.

XI. **Parapsoriasis** consists of brownish red patches, with slight or no infiltration, and fine scales. It may be very localised or widespread. Itching is usually absent. It is very resistant to treatment. The name Xantho-erythrodermia Perstans has been applied to one form which has little scaling.

GROUP II. VESICULAR AND WEEPING ERUPTIONS

Most eruptions, in which the elements are usually vesicular and the exudation serous, are commonly classed into those with small vesicles, and those with vesicles of larger size, bullæ.

I. Eczema.	XI. Pemphigus.
II. Impetigo contagiosa.	XII. Epidermolysis Bullosa.
III. Streptococcal skin infections.	
IV. Herpes.	XIII. Hydroa Aestivale.
V. Varicella.	XIV. Lymphangioma circumscriptum.
VI. Scabies.	XV. Anthrax.
VII. Tinea circinata (sometimes).	XVI. Pustular and other diseases in
VIII. Sudamina.	which vesicles and bullæ may
IX. Hydrocystoma.	occur at some stage.
	NOTE.—Syphilides are practically
X. Dermatitis herpetiformis.	never vesicular.

§ 509. I. Eczema is a catarrhal inflammation of the whole skin, running sometimes an acute, sometimes a chronic course, presenting a red excoriated surface denuded of its epithelium and more or less covered with crusts, associated in its acute stages with non-marginated swelling. " Weeping " —*i.e.*, a serous exudation which stiffens linen, is a characteristic feature. Although eczema has been defined as a vesicular disease, it may present different appearances at different stages of its course. Of recent years there has been much discussion as to the definition of eczema and dermatitis ; in the latter a cause of the inflammation of the skin which characterises both diseases is more readily detected. In eczema are seen, at different stages, the three primary and three secondary lesions of the skin—erythema, papules, vesicles, crusts, scales, and fissures. In the first stage, or *acute* eczema, there is erythema, with papules and tiny vesicles, which readily rupture, causing a serous exudation. By the time the physician sees the case—*e.g.*, in a day or two, the second or *subacute* state is usually reached, with excoriations and crusts, and the involved patch of skin presents a more or less swollen surface, often denuded of its epithelium. If the disease passes on to the third or *chronic* stage, the discharge decreases or disappears, leaving a thickened, irregular, scaly patch, fading at its margins. The fresh horny layer sheds in large scales for some time. Any part of the body may be affected, and to any extent ; but eczema has a predilection for the flexor aspects of the limbs and the flexures of the joints. The patient complains of a burning, smarting, throbbing, or itching, in proportion to the acuteness of the process.

The *Diagnosis* of eczema is not difficult. *Seborrhœic dermatitis* has no serous exudation, and is covered by greasy yellow scurf. *Syphilides* never resemble acute or subacute eczema, or, indeed, any vesicular disease, a fact of considerable value in practical diagnosis. It is difficult sometimes to distinguish patches of dry chronic eczema from *psoriasis*, but the latter affects characteristic localities, preferably the extensor aspects,

and is covered with silvery white scales.   The diagnosis of the numerous varieties will be given below.

In the parlance of to-day, eczema is due to a sensitisation of the epidermis, with reaction, caused by an external irritant or an internal toxin. Therefore history or evidence of a *cause* should be sought.   (1) *Local Causes.* —Eczema is the lesion most frequently following local irritants, such as a mustard plaster, turpentine, the leaves of certain plants (*e.g.*, the primula obconica), or soaking the hands in water containing soda.   It often occurs around the mucous orifices from which an irritating discharge issues. The local hypostatic congestion attending varicose veins is a frequent predisposing factor.   Unrecognised scabies or pediculi lead to protracted cases of eczema of multiform character.   Friction, pressure, extremes of cold and heat, cold winds, sunlight, and excessive sweating may cause eczema   Certain chemicals also cause it, as evidenced in several trades grocers (sugar), bakers (salt), washerwomen (soda), photographers (metol)   Eczema is predisposed to by very greasy and very dry skins. (2) *Constitutional Causes.*—Eczema may occur as a complication of dyspepsia, gout, diabetes, or renal disease.   In cases of eczema of the vulva or prepuce, the urine should be examined for sugar, and the presence or absence of leucorrhœa ascertained.   It often accompanies albuminuria, especially if dropsy be present.   It may appear during every pregnancy, or after the cessation of lactation.   The association of eczema and asthma has long been noticed.   When there is sensitisation to any food substance eczema may occur.   It is especially noticed with oatmeal and wheat, but may also be caused by other foods, *e.g.*, milk, pork and beef.

Varieties.—In addition to the typical acute and chronic forms described, there are several varieties of eczema.   (1) In *E. papulosum* the process stops at a papular stage.   (2) *E. pustulosum* or *impetiginoides* is due to a secondary invasion by staphylococci, occurring frequently on the heads or faces of children, on the hairy parts of delicate and tuberculous persons, or when any local irritation is present; it is attended by the formation of crusts.   (3) *E. rubrum* occurs usually on the legs, chiefly in old people. The moist red surface is often kept up by streptococcal infection. Deficient circulation produces a livid colour, and prevents repair.   (4) In *Vesicular E.* the vesicles are prominent, tend to come in crops, and become confluent.   Its favourite localities seem to be the face, the ears, and the flexures of the limbs, fingers, and toes.   Some cases of this disease resemble dermatitis herpetiformis.   (5) *E. squamosum* is a chronic scaly stage into which many erythematous and papular varieties develop, and is found most frequently on the palms, legs and scalp, and is apt to be mistaken for psoriasis and squamous syphilide.   (6) Occurring in different parts of the body, eczema is often named from the locality—E. capitis, E. ani, E. intertrigo, but it is unnecessary to invent special terms for these varieties, except, perhaps, in the case of *E. palmaris*, which is often due to the sufferer's occupation.   (7) In the palms it is apt to become chronic, dry, thickened, and fissured (*E. rimosum*)

(8) *Cheiro-pompholyx* is the term given to a vesicular and bullous eruption affecting the hands *symmetrically*, and sometimes the feet at the same time. The thickness of the epidermis in this situation prevents the rupture of the vesicles. These are especially prone to appear in the clefts between the fingers and toes, like boiled sago grains, and creep on to the palmar and dorsal surfaces—an important diagnostic feature between this disease in its later stages and a scaly syphilide of the palms. Some of the vesicles coalesce into bullæ, their contents become absorbed, and exfoliation of the epidermis occurs.

(9) *E. Marginatum* is tinea circinata of the groins and genitals (§ 502).

(10) *Paget's disease* of the nipple is really a malignant form of eczema. It starts on the nipple of one or both mammæ, and spreads centrifugally, sometimes with a slightly raised margin, leaving a reddened, congested, and sometimes weeping surface. It is met with mostly in females of advancing life, and it consists of a slowly growing cancerous process.

(11) *Peri-oral Eczema* (§ 502) is a term which was applied by the author to an epidemic condition which he observed in a large board school in 1895.

The *Treatment* of eczema differs according to the stage of the disease. First of all, all external causes should be sought for and removed, and the internal toxins carefully sought for and if possible eradicated. The principles of local treatment are those underlying the treatment of all diseases of the skin. In the acute inflammatory stages, when there is much erythema or vesiculation, rest indoors or in bed is indicated, with such soothing applications as zinc oxide, calamine, lead, or bismuth, especially in the form of lotions, powders, and pastes. Lead and weak creolin lotions are comforting. For acute eczematous conditions I have found nothing more successful than the prescription F. 42, or lot. calamin. co. Saline aperients are useful; and vin. antimon. ℥x. (0·6) with mag. sulph. ½ dr. (8) t.d., if used early, seems sometimes to cut short the disease. A daily bath (say a teaspoonful of creolin to 15 gallons of warm water) is indicated whenever the eruption is widespread, and among out-patients in whom want of cleanliness is a potent causal factor. In subacute stages a stimulating agent should be added to the soothing remedy; and in the chronic stages change of air (to hilly country in preference to seaside) and stimulating remedies. These are tar, mercury, resorcin, creosote, carbolic acid, salicylic acid, and sulphur. But a mere knowledge of the drugs to be employed is not sufficient; it is necessary to follow certain rules in their application. Thus for weeping surfaces lotions, not ointments, should be chosen; for thickened surfaces ointments should be not merely smeared on, but rubbed in or spread on a piece of lint, and firmly applied to the skin, so that a macerating effect may be obtained. The same results may be obtained and the part protected by the use of medicated plaster mulls, the most useful of which, perhaps, is a weak tar and mercury plaster (4 per cent. of the former and ¼ per cent. of the latter). It is of no use to apply remedies over thickened crusts or scabs; these must first be removed by means of bread or starch poultices, or wiped away with olive oil. When the surface is thus cleaned it must be kept aseptic by excluding the air and renewing the applications every four hours for acute, and once or twice a day for chronic, conditions.

Lotions should rarely bo covered with gutta-percha tissue to keep them moist, because tho part becomes sodden. Eczema, when acute, must never be washed with soap and water. In subacute and chronic cases bathing with warm normal saline is permitted. When ointments are used, the skin may first be cleansed with sweet oil or creolin lotion (1 in 160). In the later stages an ointment containing mercury or tar may be used (e g., F. 104), the stimulating agent being cautiously increased. If there is much secretion an astringent lotion (such as lead or creolin) may be first employed to cleanse the part, while if the part is dry and scaly the tar and mercury may bo increased without danger. In cases of long duration in which considerable thickening exists, our chief object should be to remove the products of disease so that the healthier underlying structures may resume normal growth. This may be done by the active application of exfoliating remedies such as salicylic acid (gr. 20 to 30 (1 2–2) or more) to ℥ı (32)) and sulphur. In very inveterate cases the previous application of strong plaster mulls (e.g., salicylic acid 10 to 30 per cent. and creosote 10 to 40 per cent.) facilitates the process, though it may at first appear to make it worse by setting up an acute inflammation. Constitutional treatment is specially indicated when the eruption is widespread or generalised. The stomach and intestines must bo carefully treated. Alcohol is forbidden ; tea and coffee strictly limited ; water can usually be freely taken. In some cases a lacto-vegetarian dish is indicated ; in others, restriction of the carbohydrates. The gouty diathesis and cases with septic foci present their own special features and problems of diagnosis and treatment. When there is any marked tendency to erythema or congestion three remedies are of great use—alkaline carbonates, quinine, and calcium chloride, all in fairly large doses.

Treatment of Varieties.—Eczema of the *eyelids* requires care in treating, lest the ointment should set up conjunctivitis. An ointment of hyd. ox. flav. and acid. borici, āā gr. ii. (0·13), aq. dest. and ol. amyg. dulc. āā ℔xxx, (1·8), lanolin 1 oz. (32)—is useful. For eczema around *mucous orifices*, the great indication is to keep them dry, and a powder consisting of equal parts of zinc oxide, bismuth. carb., and calamine frequently dusted on gives relief. Calamine lotion and mild mercurial ointments are also useful. X-ray and ultra-violet light give excellent results, but great care must be exercised in using them. For *E. impetiginoides* ung. hyd. am. is almost a specific ; *cheiro-pompholyx* is treated on the same lines as acute eczema ; in *E. palmaris* gloves saturated with the ointment should be worn at night, and if possible by day, and a small quantity of ointment should be smeared on after every washing ; *Paget's disease* is an indication for removal of the breast (see also § 530). Obstinate dry patches usually yield to 1 or 2 half pastille doses of X-ray.

II. Impetigo Contagiosa consists of discrete vesicles of varying size which soon become pustular (§ 515).

III. Streptococcal skin infections give rise to many different forms of vesicular eruption, which may be mistaken for eczema or herpes on the

one hand, and pemphigus or urticaria bullosa on the other. The bullæ
are situated on an inflamed base; streptococci are found inside the
bullæ.

Thè so-called pemphigus neonatorum is a pyogenic infection arising
during or soon after birth. Pemphigus contagiosus tropicus is a similar
affection occurring in the tropics in adults.

§ 510. IV. Herpes (Synonyms : Herpes Zoster, Zona, Shingles) may
be defined as an acute non-contagious disorder, consisting of one or more
clusters of vesicles on a crimson base, associated with neuralgic pain, and
due to an irritative lesion of one of the ganglia of the posterior spinal
roots or their analogue the Gasserian. Herpes commences with a red
patch or a group of flat papules, on which vesicles rapidly appear. The
vesicles are larger than those of eczema, round, hemispherical, and uniform
in size ; and as there is no tendency to spontaneous rupture there is
usually no oozing such as occurs in eczema. They smart or burn, and
the pain which precedes and follows is often very severe and intractable
The vesicles contain clear serum, and after lasting a few days, dry up
and form little crusts which fall off, leaving no ulcer. The whole attack
lasts on an average three weeks. Only rarely does deep ulceration and
scarring follow. Herpes is nearly always unilateral the groups corespond-
ing with what is known as a sensory area ; that is to say, an area which
has been shown by Mackenzie, Thorburn and Head to represent the ter-
minal distribution of the pain-appreciative fibres connected with each
spinal segment (§ 594). Many *varieties* are named according to their
position—*e.g.*, H. frontalis, ophthalmicus, cervicalis, brachialis. H.
febrilis (facialis or labialis) often accompanies inflammations of the respira-
tory tract. H. progenitalis, or preputialis, which occurs on the genital
organs of both sexes, sometimes alarms the patients with the dread of
syphilis. Serious varieties are *zoster hæmorrhagicus*, in which hæmorrhage
occurs into the vesicles, and *zoster necrogenica*, in which the skin sloughs,
and is followed by scarring or keloid. *Diagnosis.*—Herpes may be dis-
tinguished from all other vesicular conditions by the occurrence of the
vesicles in *clusters* or constellations on an erythematous base. In regard
to *Prognosis*, herpes tends to spontaneous recovery in the course of a
few weeks, except in the two grave forms above mentioned. The neuralgia
which succeeds, however, is often very intractable, especially in the aged.
Occasionally paralysis occurs, and inflammation of the structures of the
eye.

The *Treatment* is quite simple. Protect the vesicles by starch or zinc
powder, or paint with collodion, or use some soothing ointment. Quinine
in large doses, gr. 5 (0·3) three or four times a day, is reputed to be the
best remedy for the neuralgia. For *herpes preputialis* give lead lotion.
For *herpes labialis*, seek the cause in recurrent cases—infection from teeth,
tonsils, nose or colon. The vesicles may be aborted by alcohol locally,
or boracic powder.

V. Varicella. See §§ 379 and 382.

TABLE OF DIAGNOSIS.

Varicella.	Small-pox.
No symptoms before rash.	Three days before rash, sudden onset of illness with backache.
Soft pink papules becoming vesicular.	Shotty papules becoming vesicular or pustular.
Chest, neck, and trunk, fewer on face and limbs. Rash is centripetal.	First on face and wrists; more on limbs than on trunk. Rash centrifugal.
Successive crops, and thus find small papules besides vesicles of various sizes.	All one stage (papular or vesicular, or pustular) at one place.

VI. **Scabies** is chiefly a papular eruption (§ 496) ; but in children the vesicular element often predominates ; it may then be mistaken for a septic eczema or varicella. The burrows, and the tendency of scabies to affect the positions where the skin is soft should aid the diagnosis.

§ 511. VII. **Tinea Circinata**, or ringworm of the body, is occasionally vesicular, especially when the fungus is of animal origin, the arrangement of the vesicles in the form of a definite ring being so characteristic as to be unmistakable (Fig. 130). The usual naked-eye appearance of this lesion is a pale red ring with a scurfy margin. With a lens the margin is seen to be slightly raised with minute papules or vesicles. When

FIG. 130.—Hand of a woman suffering from TINEA CIRCINATA, in which the vesicular element is unusually prominent. A large ring of vesicles encloses a scurfy area. Verified by microscopic examination.

originating from the horse, there may be distinct suppuration. The favourite localities are the face, neck, and arms. When occurring in the genito-crural region, it used to be called eczema marginatum (see § 502). It is the most common cause of the so-called eczema between the toes. This form is associated with recurrent attacks of streptococcal lymphangitis spreading up the leg. Under the microscope, the mycelium (Fig. 131), and perhaps a few spores of the tricophyton (the large-spored fungus, § 529) can be seen in scrapings from the roof of vesicles or the scales from the margin of patches. The treatment consists of rubbing in ung. hyd. amm. chlor., or some other parasiticide, such as Whitfield's combination of salicylic and benzoic acid, as 1 to 24 parts of soft paraffin.

§ 512. VIII. Sudamina are clear, scattered, non-inflammatory vesicles, like droplets of water, about the size of a pin's head, occurring in conditions such as acute rheumatism, which are attended by very profuse perspiration. They give rise to no inconvenience, and disappear in a few days. They are a non inflammatory disorder of the sweat glands, whereas in *miliaria* (commonly called prickly heat) a mildly inflammatory condition of these glands gives rise to similar papules and vesicles on a red base

IX Hydrocystoma is a rare disease characterised by deep-seated, tense, translucent vesicles occurring on the face, varying in size from a pin-head to a pea, very persistent, lasting for months. They are formed by a cystic swelling of the duct of the sweat-gland, and never become purulent They disappear. spontaneously, chiefly in cold weather. The disease occurs chiefly in middle aged women, especially in those whose life is spent in a warm, moist atmosphere, or who perspire much. It frequently comes on as warm weather sets in The *Treatment* consists in puncturing the vesicles

§ 513. X **Dermatitis Herpetiformis**[1] was described by Duhring and may be defined as a relapsing disorder of prolonged duration, characterised by the appearance of successive crops of erythematous or papular elements, always in clusters, which usually go on to the formation of vesicles, pustules, or bullæ, are always attended by intense irritation, and sometimes by pigmentation. Different varieties are described according to the element which predominates In some (the *erythematous* variety) the preponderating eruption consists of circumscribed patches of bright red
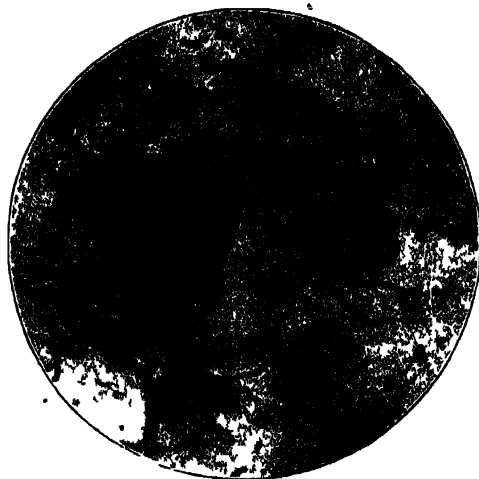

Fig 131 — Mycellium of TINEA CIRCINATA (ringworm of the body) —A scraping from the skin, stained by Gram's method Mycellium of equal segments having truncated ends bifurcating in places; spores almost absent Compare Fig, § 529

erythematous or semi-urticarial inflammation, which spread by raised edges, and leave a pigmented centre In the usual variety (the *papulo-vesicular*), vesicles which vary from the size of a pin's head to a split pea predominate. Sometimes these vesicles become bullæ as big as a walnut (the *bullous* variety). There is acute inflammation of the deep layer of the skin. The fluid in the bullæ contains eosinophil cells in great excess, and this is true also of the blood. Sometimes the predominating element is a papular one, and because of the itching the heads of those become scratched and covered with blood-stained scabs. The intense itching is a very notable feature in all the varieties. Scars and temporary pigmentation may ensue. In many cases the general health seems undisturbed, but often the appearance of each crop is attended by pyrexia, and occasionally gastro-intestinal disturbance. The distribution tends to be symmetrical, and to favour the flexor surface of the wrists, the axillæ, groin, abdomen, and buttocks. The mucous membrane of the mouth and pharynx may

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[1] A few of the synonyms of Dermatitis Herpetiformis : herpes gestationis (Milton 1872) ; hydroa gestationis (Liveing) ; hydroa herpetiforme ; pemphigus arthritique (Bazin) ; impetigo herpetiformis of Hebra ; dermatite polymorphe prurigineuse, ou douloureuse, chronique à poussées successives (Brocq).

also be involved. Each successive crop lasts from one to four weeks; each attack being separated by longer or shorter intervals of comparative freedom. In this manner the disease may go on for months or years.

Diagnosis.—The disease differs from pemphigus vulgaris in the following respects : (1) The smaller size of the vesicles or bullæ, which are (2) constantly arranged in clusters; (3) the presence of erythematous patches beneath the vesicles and elsewhere, and the multiform character of the eruption ; and (4) the presence of itching. From *eczema, urticaria bullosa,* and *erythema multiforme* the disease is distinguished by consideration of the above features.

Etiology.—The disorder is more common in men than in women, and between the ages of sixteen and thirty. It is supposed to be due to a poison of intestinal origin.

Treatment.—Arsenic is of great service, and should be given in full doses. The disease is very obstinate. Quinine and salicin for the irritation are useful. Locally, sedative lotions and ointments may be prescribed, much the same as those recommended in acute eczema (*q.v.*), while Duhring advocated sulphur ointments, ʒii. (8) in ʒi. (32), and tar lotions. Lumbar puncture sometimes relieves the itching.

§ 514. XI. **Pemphigus** is one of the rarer diseases of the skin, characterised by the presence of bull o and constitutional symptoms. *P. chronicus* or *vulgaris* is the more common and typical variety in which the bullæ develop in crops, each bulla varying in size from a pea to a hen's egg, being tense with clear albuminous serum, which becomes turbid, purulent, and occasionally hæmorrhagic (P. hæmorrhagicus). The bulla is characterised by having at first no ring of erythema round its base. The fluid is either absorbed with formation of crusts, or the blebs burst, leaving a raw surface on which new epidermis soon develops. Almost any part of the skin may be affected, as well as the mucous membrane of the mouth, nose, throat, vulva and conjunctivæ. Each bulla only lasts a few days ; fresh crops may continue to come out for months. The constitutional disturbance depends largely upon the number of bullæ and the frequency of the crops. Many cases are apt to recur throughout life. The outlook is grave ; even after long intervals of comparative freedom the issue is fatal in practically all cases. In *P. foliaceus* the bullæ are very thin and flaccid, and rupture early ; but the epidermis, instead of re-forming, continues to peel off until large areas of red, raw, exuding surface are exposed, with epidermis folded at the margins—a point which distinguishes it from eczema rubrum. This process slowly extends for a year or two until the whole body may be involved, and a fatal issue ensues. *P. vegetans* is a rare variety, which develops papillomatous vegetations. Starting in the mouth, the disease spreads ; the bullæ remain long unhealed, and in moist positions such as the groins and axillæ vegetations develop with offensive discharge. Prostration and death usually occur in a few months. *Etiology.* — The cause of pemphigus is unknown. The *treatment* is palliative. Arsenic in gradually increasing doses in some cases does wonders. Quinine and other tonics are called for, especially in cachectic cases. Transfusion aids some cases. Morphia is required towards the end. Among local remedies the continuous bath in severe cases, and dusting powders or boracic ointment to protect from septic influences in milder cases are the best.

XII. **Epidermolysis Bullosa** is a rare congenital disease in which slight traumatism causes the formation of bullæ. It usually runs in families. The bullæ appear on parts exposed to friction or pressure, and even the nails are affected. I have found ergot useful.

XIII. **Hydroa Aestivale** is a papulo-vesicular or bullous eruption occurring on parts exposed to the action of light, and therefore appears chiefly in summer. Scars frequently follow the bullæ. In the juvenile type it is often associated with hæmatoporphyrinuria and is due to an inborn error of metabolism. The adult type is less severe and hæmatoporphyrinuria disappears on a meat free diet ; the gastric acid is low in these cases (H. W. Barber).

XIV. **Lymphangioma circumscriptum** is due to dilatation and overgrowth of lymph vessels, and causes an appearance of deep-seated, thick-walled yellow vesicles.

XV. **Anthrax begins as a** papule. A ring of vesicles forms, and on an inflamed base a central gangrenous scab develops. Constitutional symptoms are present.

XVI. The description of the other diseases occasionally characterised by vesicles or bullæ—erythema multiforme, urticaria, drugs, congenital syphilis, leprosy, etc., will be found under their respective headings. Stings and bites of insects may cause small or large vesicles, with a central punctum which aids diagnosis in obscure cases.

GROUP III.   PUSTULAR ERUPTIONS

Eruptions in which the elements are mainly pustular naturally fall into three classes—

(a) *Superficial Pustules.*

I. Impetigo contagiosa.
II. Ecthyma.
III. Impetigo Herpetiformis.
IV. Cutaneous Diphtheria.

(b) *Pustules on an Indurated Base.*

V. Pustular syphilide.
VI. Sycosis.
VII. Pustular acne.
VIII. Pustular folliculitis.

IX. Bromide and other drug eruptions
X. Variola
XI. Acute glanders
XII. Pustular tuberculide

(c) *Furuncular eruptions with a Slough.*

XIII. Boils.
XIV. Carbuncles.
XV. Kerion.
XVI. Anthrax in its later stages.

Hebra wrote in 1870 " all pustules must be regarded as secondary morbid products, and hence are not fitted to form an independent series of cutaneous diseases." Curiously enough almost the only exception to this dogma is to be found in that rare condition Impetigo Herpetiformis, which was first described by Hebra. *Eczema and all the diseases mentioned in Group II may become pustular,* owing as we now know to infection by pyogenic cocci. Conversely, nearly all the pustular diseases just mentioned may start as vesicles.

According to Dr. Sabouraud there are three prevailing cocci found in the skin. (1) The streptococcus which is the cause of the impetigo contagiosa, and for this he recommends 1 or 2 per cent. zinc sulphate ; (2) staphylococcus aureus, which invades the follicular orifices and causes pustules, being the cause of all primary and secondary pustular lesions, and for this he recommends sulphur 19 grammes, alcohol 30 grammes, aq. rosæ 100 grammes ; and (3) staphylococcus griseus, which causes pityriasis steatoides and seborrhœic dermatitis. For this he recommends oil of cade 10 grammes, yellow binoxide of mercury 1 gramme, and pet. moll. 30 grammes.

(a) Superficial Pustules

§ 515. I. Impetigo Contagiosa (Synonym : Porrigo) is frequently met with on the faces of children, and is so called because it is readily conveyed from one child to another. At first the spots are vesicular, but they become pustular in a few hours. The pustules vary in size, and are discrete, but may run together if near each other. In the course of a few days they dry into yellow crusts, which, falling off, leave a flat congested mark covered by new cuticle. They do not leave scars unless scratched. The favourite positions are the face, especially round the mouth, scalp, and hands of children, but they may occur on any part of the body. If

untreated, fresh pustules appear in other places for a week or two ; or the disease may die out spontaneously in a few weeks. It is usually trivial, without constitutional disturbance, and with only slight itching. The disease may be conveyed by contact to other parts of the same or to another individual.

Etiology.—Impetigo will spread through a school (*e.g.*, scrum-pox in football) or family of children, attacking weak and strong alike. Adults enjoy remarkable immunity, but occasionally contract it from a child. The essential cause is a streptococcus just below the horny layer, soon followed by staphylococcal infection.

Diagnosis.—Impetigo pustules are readily distinguished from acne, sycosis, pustular syphilide, and all other pustular eruptions by (i.) their superficial character, and (ii.) their typical localities. Severe forms of bullous impetigo are the same as the so-called pemphigus neonatorum, and may become fatal. A *circinate* impetigo may form gyrate patches.

The *Treatment* is extremely simple. The crusts must first be removed with warm water, a little sweet oil, or a starch poultice ; and then a few applications of sublimate solution, or ung. hyd. am. chlor. are sufficient. Mercury is almost a specific in pustular affections.

II. **Ecthyma** is a term used to describe *large isolated pustules*, or the superficial sores which form part of impetigo, scabies, pediculosis, etc., in children and aged persons broken down in health, and wanting in personal cleanliness. They may have to be distinguished from scabies in children and from suppurating syphilides.

III. **Impetigo Herpetiformis** is a rare disease described by Hebra. It is characterised by the appearance of clusters of miliary pustules, usually starting on the inner surface of the thighs, whence they spread, generally associated with the pregnant or puerperal state, and usually terminating fatally. The tongue, palate, pharynx, and even the œsophagus have been the seat of pustules and superficial ulceration. There is considerable fever of pyæmic type, and each fresh crop of pustules is attended by rigors and increasing prostration. Most cases have been connected with the later months of pregnancy, and if the patient recovers from the first attack the disease seems to be apt to recur in a subsequent pregnancy.

The *Treatment* should be conducted on the lines of other septicæmias. Locally, mild mercurial lotions and ointments can be given. Autogenous serum injections have done good.

IV. **Diphtheria of the skin** resembles a widespread, obstinate impetigo, with large, sometimes sanious crusts, and is usually diagnosed by the discovery of the microbe after the disease has resisted ordinary treatment for impetigo. Injections and local applications of diphtheria antitoxin readily cure the lesions.

(b) Pustules on an Indurated Base

§ 516. V. **Pustular Syphilides** are of two types : (1) *Small Papulo-pustular Syphilide* (Acneform Syphilide, or Lichen Syphiliticus Pustulosus) consists of spots about the size of a pin's head, upon a hard base, which in a week or ten days scab off, leaving the characteristic indurated papules with depressed centres. They are arranged in groups, circles, or circular lines. (2) *Large Pustular Syphilide* (Rupia, Ecthymatous Syphilide, Variola Syphilitica) consists of pustules varying in size from a split pea to a halfpenny, flat or hemispherical, and surrounded by a raised brick-red

infiltrated margin. They may be grouped, ringed, or isolated. The pustule bursts, the pus escapes, and crusts are formed with ulceration beneath them. The ulceration tends to spread serpiginously, and leaves permanent scars, rings, and pigmentation. Both varieties may occur on any part of the body, and both indicate a malignant type of syphilis. The smaller pustular syphilide may have to be *diagnosed* from *acne* by the presence of comedones, and the slower course in *acne*. When on the face pustular syphilide may be hard to distinguish from *lupus vulgaris*, but the youth of the patient in the latter complaint, and the extremely slow rate of progress, aid us. When pustular syphilide is diffuse, it may be mistaken for *variola*, but in the latter there is a history of a vesicular stage, of backache, and constitutional symptoms.

§ **517. VI. Sycosis** is a term applied to a slowly growing pustular eruption, evidence of a deep staphylococcal infection of the sebaceous glands and hair follicles of the beard and sometimes the moustache. Indurated papules at first appear, and many undergo suppuration. Clinically there are three conditions which present the appearance which we describe as sycosis ($\sigma v \kappa o v$ = a fig). (1) True Sycosis (Synonym : Folliculitis Barbæ) in which the pustules become large and indurated, and in this later stage the hairs can be easily drawn out, followed by a drop of pus. The condition is due to the staphylococcus aureus ; hence it is sometimes called coccogenic sycosis. It may be contracted at the barber's, or it may be due to a nasal discharge. (2) Tinea Sycosis (Synonym : Hyphogenic Sycosis) is ringworm of the beard due to the trichophyton tonsurans. The large spored ringworm in children, and the ringworm of horses, cows, cats, and dogs may produce this disease. There are two varieties : (*a*) *Superficial*, characterised by scaly red rings, in which the hairs are only slightly involved ; and (*b*) *deep-seated*, in which hard nodules and lumps are formed with broken hairs. In this form the hairs are easily pulled out from the onset. (3) *Eczema barbæ* may become secondarily infected. In true sycosis the pustules are the predominating element, and the intervening inflammation is secondary. In eczema barbæ the eczematous condition affects the intervening parts, and spreads on to the face as well. All three conditions may, if untreated, last for a considerable time.

Unless the trichophyton be found, it is often very difficult to say which condition is present. In *impetigo contagiosa* the pustules and crusts are quite superficial and readily distinguished from sycosis. The *Treatment* is prolonged. The hair should be kept short. Spirit and mercurial lotions suit many cases. Salicylic and carbolic acids and mercury are, in my experience, the best reagents to use. The pustules should be opened. In some cases vaccines, especially when autogenous, bring about a rapid cure ; in others, epilation by means of X-rays is successful.

§ **518.** Various other pustular eruptions may be mentioned :

VII. **Pustular Acne** is recognised at once by the presence of comedones, papules, and pustules on the face, and sometimes the upper part of the back. This and the severe pustular form known as acne varioliformis is described in § 404.

VIII. **Pustular Folliculitis** is a papulo-pustular condition specially affecting the hair follicles, due to infection by the staphylococcus aureus. It affects the hairy parts, especially the legs in men. It may be distinguished from a syphilide by the fact that each pustule involves a single hair follicle. In **Bockhardt's impetigo** only the upper third of the pilo-sebaceous follicle is involved. It is a superficial staphylococcal infection occurring chiefly on the scalps of children, or on sites where an irritant has been rubbed into the skin.

IX. **Iodides** and **Bromides** sometimes produce pustular eruptions on a congested indurated base. Antimony, aconite, arsenic, sulphide of calcium, nitric and salicylic acids also produce pustular eruptions.

X. **Variola** (Small-pox).—The concluding stage of the eruption in this infectious fever is another illustration of pustules forming upon an indurated base (§ 382).

XI. The eruption of **Acute Glanders** when it has reached a pustular stage is so much like small-pox that it may very pardonably be mistaken for it (§ 389).

* XII. A pustular **Tuberculide** may appear as small or large pustules arising on a hard papular base. These may coalesce, and under the scab an ulcerating surface develops.

(c.) *Pustular Eruptions prone to become Furuncular, or Sloughing :
viz., Boil, Carbuncle, and Kerion*

§ 519. XIII. A **Furuncle,** or boil, is an acute circumscribed, suppurative inflammation in the skin, varying in size from a small pin's head to a bean. When the process fails to pierce the skin it is called a *blind* boil. As a rule, however, the inflammation involves the surface, which breaks and permits, in the course of a few days, of the discharge of the central necrosed portion, which is spoken of as the core. The cup-shaped cavity which is left heals by granulation, and a scar remains. The pain is considerable, especially in the early stages, and varies with the tension of the part. The staphylococcus aureus is the usual causal organism. *Furunculosis* is the term applied to the condition in which boils are constantly recurring over a prolonged period at different parts of the body.

Treatment.—Protect the part from external irritation. Incision is usually necessary. Stannoxyl, collosol manganese, and dilute sulphuric acid in ♏xx doses t.i.d. are useful, and the general nutrition should be improved. Other cases do better with alkalies. In cases of recurring boils, examine the urine for sugar; seek for and remove any infecting focus. I have found this in some cases in the teeth; in others in the colon. Vaccine treatment (beginning with 50 million staphylococcus aureus) is very successful. High-frequency currents, ultra-violet light and pure ichthyol may abort boils.

XIV. A **Carbuncle** may be regarded as a combination of several boils side by side, constituting an inflammatory area of considerable size spreading beneath the skin, with numerous openings in the skin through which the pus pours. A leathery slough forms as it were a sheet in the deeper layers of the derma. Its commonest position is the neck or back, but it may occur on the sacral region from pressure, on the face (when a neurotic element can generally be traced), or elsewhere. The pain and constitutional disturbance are often very severe, and if the carbuncle be extensive general pyæmia may ensue, or death from exhaustion. The *Diagnosis* is never difficult, on account of the characteristic red, infiltrated, swollen, circumscribed area in one of the positions named, and honeycombed appearance with pus-discharging holes. The *Causes* are much the same as those of boils, though carbuncle is more often the result.

of debilitated states and diabetes, and is more often found in the aged suffering from cardio-vascular disease.

Treatment.—Warmth is in my experience most useful to check the extension, or, if this is impossible, to promote suppuration, and the separation of the slough. When by unmistakable fluctuation we know that suppuration has ensued, free crucial incisions should be made, the slough cut away as freely as possible, and frequent syringings every hour or so with a carbolic lotion, 1 in 100, adopted. Iron, arsenic, strychnine, and a liberal diet should be freely administered. Vaccines are not so useful as for boils, but should be tried. Zinc and copper ionisation hasten healing.

§ XV. Kerion is a condition occurring chiefly on the heads of children suffering from ringworm, due usually to an ectothrix infection, of animal origin. Occasionally it may also be seen in cases infected with small-spored ringworm. Superficially it resembles a carbuncle, but without the same induration. It is a circular, raised, inflamed, boggy area of skin through which are a number of pus-discharging holes (see § 529).

GROUP IV. MULTIFORM ERUPTIONS

Multiform eruptions are sometimes found in the following conditions – syphilis, scabies, eczema, erythema multiforme, varicella, leprosy, and dermatitis herpetiformis.

§ 520. General Characters of Syphilitic Eruptions. —Syphilitic eruptions have already been referred to under Syphilis (§ 434) and under papular and scaly eruptions. (1) They are of many different *kinds*, and several kinds may be present at one time (polymorphism). All kinds of elementary lesions may appear on the skin with the single exception of vesicles ; eczema and other vesicular lesions are never found as a result of syphilis —a diagnostic feature of great importance. (2) The syphilitic *papule* may be regarded as a prototype of a syphilitic skin lesion. It is the starting-point of them all. (3) The *features common* to all syphilitic rashes are their reddish-brown colour, generalised or symmetrical distribution, grouping in segments of circles, preference for the forehead and flexor aspects, polymorphism and absence of itching. The later skin lesions in malignant cases (in which a so-called tertiary stage occurs) differ, however, in being asymmetrical, and with a marked tendency to ulceration.

The clinical features which distinguish syphilides are explained by three histological facts. (1) All syphilides are due to a deposit in the dermis or epidermis of a cellular *infiltration*. Hence the colour does not disappear on pressure, and is followed by staining. (2) The cells constituting this gummatous or granulomatous infiltration are of low vitality. They do not organise into connective tissue, but tend to undergo either *involution* by absorption on the one hand, or *suppuration* and pustulation on the other. Hence the depressed cup-shaped centre, and the great tendency to polymorphism. Hence also the absence of vesiculation or an eczematous form of eruption. (3) The infiltration *spreads centrifugally*. Hence the raised peripheral edge is the newest part, the shape most frequently assumed being that of a crescent, circle, or segment of a circle, leaving a stained centre where the papule began. If these three principles be appreciated all the clinical features are explained.

Scabies (§ 496), as it occurs in children is nearly always a multiform eruption, consisting of papules, vesicles, sometimes pustules, scratch-marks, and burrows. By the presence of the latter and the position of the eruption the diagnosis is arrived at.

GROUP V. NODULAR ERUPTIONS AND TUMOURS OF THE SKIN

A nodule may be defined as a solid deposit in the skin, which is larger than a papule. The common forms are: I. Lupus Vulgaris; II. Syphilitic Gummata; III. Various Benign Tumours (e.g., sebaceous cyst, lipoma, rheumatic nodules, vascular naevi, etc.); and IV. Epithelioma; while the rarer forms include: Leprosy; Basin's Disease; Molluscum Contagiosum; Molluscum Fibrosum; Sarcoma; Actinomycosis; Leukæmia; Yaws; Mycosis Fungoides; Blastomycosis; Sporotrichosis; Delhi Boil; Xanthoma Tuberosum; Myoma Cutis; and Madura Foot.

Some eruptions usually papular may take on a nodular form—e.g., urticaria pigmentosa, congenital xanthoma (§ 526).

§ 521. I Lupus Vulgaris may be summarily defined as a chronic disease of the skin, characterised by a collection of reddish-brown, semi-translucent (" apple-jelly ") nodules embedded in the corium, which give rise to some general thickening and desquamation, and have a tendency to ulcerate and to result in cicatricial atrophy. Their favourite position is the face, in which position the patches are rather unsymmetrical, as in l. erythematosus. They occasionally affect the hands in 20 per cent. of the cases according to Kaposi. The disease almost invariably starts early in life —in childhood. Sometimes it is found extensively over the body, its onset dating usually from an attack of measles or other acute specific fever.

The Prognosis of lupus vulgaris turns principally on three things: (1) Its position, (2) its extent, and (3) the general condition of the patient. Untreated, the disease will spread for years.

Treatment.—General hygienic and tonic measures as for phthisis are useful—e.g., good food, fresh air, malt, and cod-liver oil. Prolonged exposure to bright sunlight when available is curative. Failing sunshine, the local areas clear up when the entire body pigments after exposure to artificial sunlight. Cod-liver oil and iodides act wonderfully well in certain cases; tuberculin injections are used when the disease is widespread over the body. Local treatment consists in (1) Diathermy and high-frequency fulguration; (2) curetting, followed by caustics or the cautery, the result of which is often very satisfactory in a localised patch, but unfortunately in most cases requires periodic repetition; (3) Finsen lamp (photo-therapy), which is best suited to limited patches and yields excellent results; (4) ionisation; (5) caustics and escharotics like acid nitrate of mercury carefully applied, carbolic and salicylic acid, etc., are extremely useful. Dr. Adamson's method of application of the acid nitrate of mercury is very efficacious in many cases. When the mucous membrane is involved Pfannenstiel's method is good: locally, swabs of hydrogen peroxide, and internally half-drachm doses (2) of sodium iodide.

II. Syphilitic Gummata occur in the skin for the most part in the later stages of the disease. They are met with as round or ovoid nodules in or beneath the skin. In the course of a few weeks they usually make their way to the surface in the form of an indolent abscess, which leads

TABLE OF DIAGNOSIS.

Nodular Syphilide.	Lupus Vulgaris.	Lupus Erythematosus.
Nodular or diffuse infiltration with raised edges.	"Apple-jelly" nodules in derma. Sebaceous follicles not specially involved.	Superficial erythema. Sebaceous follicles plugged with hard sebum.
Destroys more in a month than lupus in a year. Stellate scarring.	Destroys slowly and usually leaves puckered scar.	Never ulcerates, though may leave a superficial scar.
Sometimes symmetrical.	Asymmetrical.	Bat's-wing distribution on face. Generally symmetrical.
Adults.	First appears in childhood.	First appears in middle life.
Amenable to Hg and KI.	Hg and KI do harm.	Hg and KI no good.

a circumscribed punched-out ulcer, sometimes of considerable depth. They may occur anywhere, but especially on the legs, brow, nose and sterno-clavicular region. They should not be lanced.

III. There are several other relatively common **Benign Tumours** or nodules originating in the subcutaneous tissue, which may involve the skin—*e.g.*, sebaceous cyst, fatty tumour, rheumatic nodules, fibro-neuroma, subcutaneous nævi, and lymphangiectasis. **Sebaceous Cyst** (Synonyms: Steatoma, Wen) is a tense, painless, cystic tumour due to the occlusion of a sebaceous follicle sometimes associated with acne, and usually single. For its eradication the *capsule* must be entirely destroyed. **Fatty Tumours** are known by their doughy feel, lobulation, the puckered depressions seen on trying to lift up the skin over them. **Rheumatic Nodules** occur in successive crops, as small, hard, or elastic nodules, sometimes adherent to the skin, usually freely movable beneath, sometimes tender on pressure. Their favourite situation is over the fibrous tissue of the superficial bones—that is to say, chiefly around the joints and along the spine.

IV. **Epithelioma,** epithelial cancer, affects the skin in several forms, and the chief site is the face. 1. In the *papular* form it is found as hard, glistening, pale, flat papules, which grow very slowly, become cracked, fissured, and ulcerated (*vide* Ulceration). 2. The *nodular* or deep-seated form is less frequently met with. It occurs as close-set, flat, or slightly raised, "very firm and somewhat translucent nodules. In the course of months or years it grows into a spherical or flat, hard tumour, whose surface is shining, waxy, or rosy, traversed by vessels, irregularly nodular. As the result of spontaneous reaction the centre is often drawn in like an umbilicus; the edges are steep and smooth" (Kaposi). Later, ulceration occurs. 3. Papillomatous or warty growths. 4. *Bowen's disease*, of squamous cell origin, is rare. The first three may be found in the same individual; the first is the most common and slow growing. The favourite sites are the lower lip—at least 50 per cent,—the tongue, and external genitalia. It may occur on a scar, on old lupus patches, or on a senile wart. The majority of cases occur in men. Lesions having these features occurring in a person past middle life should always be examined microscopically.

In the late stage of Xeroderma pigmentosum, scars of lupus vulgaris and X-ray dermatitis, malignant growths often develop.

§ 522. Certain rarer forms of nodule and neoplasm also affect the skin.

Leprosy (Synonyms: Lepra, Elephantiasis Graecorum, Leontiasis Satyriasis) is a chronic constitutional disease, characterised by pigmentary, sensory, and nodular changes in the skin, due to a specific microbe affecting the skin and nerves. Leprosy used to be a widely prevalent disease, but only imported cases are now found in England. It is still endemic in Norway, parts of Russia, Turkey, and the Turkish provinces, and in China, India, West Indies, etc. Sir Jonathan Hutchinson believes that the infection is conveyed by fish. It is communicable from man to man, though its infectivity is feeble, and probably only through an abrasion of the surface. The disease is met with in two clinical forms in its earlier stages. (a) *Maculo-anæsthetic* leprosy, which consists of patches of anæsthesia, sometimes of pigmentation or leucoderma, usually associated with thickening of the nerve trunk connected with the part, and a widespread eruption of reddish spots and patches over the body. These signs may be preceded by pain, and followed by paralysis and atrophy of the muscles supplied by the affected nerves. (b) *Nodular* leprosy, in which are found small diffuse thickenings, sometimes pink, yellowish-brown, or without much alteration of colour of the dermal tissue and mucous membranes. These increase to form bosses, and occurring on the face give to the patient a leonine aspect in course of time (facies leonis). The viscera and mucous membranes are similarly involved, and wherever the granulomatous material is formed the characteristic bacillus is found, which closely resembles the bacillus of tubercle. *Mixed* forms of these two types are met with. The course of the disease is extremely prolonged, and generally fatal. Any age may be affected. It is endemic in certain countries, where want of cleanliness and hygienic principles lead to the transmission of the disease from person to person.

Treatment.—Chaulmoogra oil, 200 to 300 minims in capsules per diem, arrests the progress when given in gradually increasing doses. It is so nauseating that it is often administered hypodermically (60 c.c., camphorated oil 60 c.c., resorcin 4 gm.), or in the form of sod. gynocardate gr. 4 in 1 c.c. water, or intravenously, gr. $\frac{1}{10}$ to $\frac{1}{2}$, twice a week.

Erythema Induratum Scrofulosorum (Bazin's Disease) is a rare tuberculide, affecting chiefly young strumous women, and characterised by chronic subcutaneous nodules in the calves of the legs, which may ulcerate. They are sometimes difficult to distinguish from syphilitic gummata, but the latter are much more rapid in their progress, and yield to iodides. A *spurious form of Bazin's disease* is met with in young persons who have a feeble circulation. This leads to hypostasis of the legs, the skin of which is apt to be thickened and livid at certain spots, but the subcutaneous nodules of Bazin's disease are wanting.

Molluscum Contagiosum consists of rounded, pearl-like elevations, varying in size from a pin's head to a pea, and semi-translucent appearance. A tiny depression is found in the centre through which the contents can be squeezed. If left alone inflammation and suppuration may occur, with spontaneous cure. The treatment consists either in snipping them off, or in squeezing out the contents, and touching the inner surface with silver nitrate or iodine.

Molluscum Fibrosum is a rare condition which consists in the formation of fibrous tissue in the deeper layers of the corium, slowly developing into tumours of varying size (up to 32 pounds), which may be sessile or pedunculated. Their favourite situation is the back. They should be removed by knife or ligature. One case under my care improved by painting with ethylate of sodium. In *Von Recklinghausen's disease* there are pedunculated growths containing nerve as well as fibrous tissue; and pigmentation occurs in patches.

Sarcoma Cutis may occur either as a primary affection or secondary to deposits elsewhere. It is met with in the form of purplish tumours of varying size of hard or spongy consistence. A small deposit with satellites around it is very characteristic,

Sarcoma may develop on pigmented moles, a melanotic sarcoma being then reproduced elsewhere.

Actinomycosis is a rare chronic affection of the subcutaneous tissue, usually starting in the jaw, and spreading thence to the skin of the face and neck. It is due to the ray' fungus or Actinomyces, which gives rise to a hard, slow-growing tumour, going on to ulceration, with a thin sero-purulent discharge, containing *yellow granules in which the ray fungus can be found* (§ 667). In other cases the disease attacks the lungs, the digestive tract, or the liver, causing much constitutional disturbance. The fungus enters through eating diseased grain improperly cooked. It has been detected in imported ox tongues. The prognosis is favourable if the disease is on the surface, where it can be dealt with surgically, or if treated early with large doses of pot. iod.

In Leuksemia and in lymphadenoma there are in a few cases nodules in the skin of the same character as those in the spleen, liver, etc. These vary in colour from that of the surrounding skin to a deep red or even to a distinct grey. They are not infrequently the site of hæmorrhage. The greenish hue of an old bruise may give rise to the suggestion that they are chloromatous, but this is seldom the case. They appear in any position, and are variable in size and persistence, sometimes disappearing for months at a time. If a blood examination is not made, such cases are often regarded as mycosis fungoides. Itching may be severe or absent.

Framboesia or Yaws is a chronic disease endemic in the tropics, rarely attacking the white population. The commonest form of eruption has a fungoid or raspberry-like character, whence its name is derived. Some observers consider the disease as identical with syphilis, modified by the climate, but it has been inoculated into syphilitic patients. It is readily cured by salvarsan.

Delhi Boil (Bagdad button, Oriental sore, cutaneous Leishmaniasis) has come from the East with the troops. It begins on the exposed parts of face and hands as a hard papule, which enlarges, softens, becomes dark purple in hue, and in three or four months usually ulcerates, with a foul yellow secretion. It tends to heal, leaving a scar in about a year. The diagnosis is made by finding the protozoal organism in the papule or ulcer margins. Locally a pastille dose of X-ray or scraping and 2 per cent. tartrate of antimony ointment are good. Cure is often obtained by intravenous injections of tartar emetic, 1 per cent. in 10 c.c. water every second day. Espundia is a similar condition found in S. America. It is accompanied by oro-pharyngeal ulceration, and is amenable to tartar emetic injections.

Mycosis Fungoides is a rare condition, characterised by the formation, after a long preliminary period, of reddish fungoid tumours. In the preliminary stages, which may last for months or years, there is an erythema or a scaly eczema attended by itching, followed by brownish-red papules, which leave pigmented and atrophied depressions, and are finally followed by smooth purple tumours, sessile or pedunculated, which ulcerate, with a typical granulomatous base. ' The eruption usually appears on the trunk, and leads to emaciation and death. X-rays have proved useful.

Blastomycosis is an extremely rare disease affecting chiefly the face and hands, characterised by papillomatous ulceration simulating tuberculosis cutis, and due to the fungus blastomyces. It may attack the viscera primarily or secondarily. Iodides and X-rays are useful.

Sporotrichosis is a rare disease due to the presence of sporotrichia, which apparently reach the skin through small abrasions. Inflammatory nodules occur on skin and mucous membranes, in subcutaneous tissue and bones. Some ulcerate and discharge a sticky pus in which the organism can be discovered. It may cause difficulty in diagnosis owing to its clinical resemblances to tuberculosis, syphilis, Bazin's disease or boils. The disease yields to treatment with iodides.

Xanthoma occurs with liver disease and diabetes in the form of raised yellow patches (X. planum) or nodules (X. tuberosum). (Cf. p. 733.)

Myoma is very rare. The smooth soft nodules contract with cold.

Madura Foot is a granulomatous condition due to streptothrix and other infection. It attacks the foot, appearing first as nodules with bullae, which break down, exuding

granular masses. The whole foot becomes swollen, the leg above atrophies. Surgical intervention is necessary. The disease occurs in India, Africa, and America.

GROUP VI. ULCERATIONS

§ 523. An ulcer is a loss of substance of the dermis and epidermis exposing a granular surface, which secretes a sero-purulent fluid. Ulcers must not be confused with large vesicular or bullous lesions, such as occur in pemphigus foliaceus, in which the skin is only denuded of its cuticle. For clinical purposes ulcers may be divided into four groups :

(a) *Idiopathic* or *inflammatory ulcers*, caused by injury, cold, hypostatic congestion, or varicose veins, and often aggravated by B. coli or other infection, gout, anæmia, or scurvy.

In the *Treatment* of chronic ulcers the main point to remember is their absence of tendency to repair. (1) Many *local applications* have been tried. If the discharge is watery and excessive, and the granulations turgid, astringents are called for, such as zinc sulphate or lead lotion, or the painting on of nitrate of silver, gr. 20 (1·2) to the ounce (32), or the use of the solid stick, which acts also as an excellent stimulant. For the troublesome itching, carbolic lotion, 1 in 50, or 1 in 100, freely applied on lint or rag, often gives relief. Various protective dressings, such as strapping and Unna's Gelatine, are often successful. (2) Rest in the horizontal position is more efficacious than any other kind of treatment for ulcer of the leg, because the deficient return of the blood is one of the factors which prevent repair. With the same object a Martin's rubber bandage, a flannel roller or an elastic stocking is advantageous. (3) A liberal diet, and a moderate use of stimulants are often successful, combined with tonics, and among the internal remedies which I have found successful with aged persons is tr. opii, m. 2 to 5 (0·1–0·3), thrice daily. It acts by improving the tone of the cutaneous vessels. It may be combined with strychnine, one of the best tonics for the aged. Calcium lactate I have used for many years. Recently Vines proved that parathyroid aids its action and, in some cases, is most successful. (4) Incisions may be made through callous, indolent edges, either at right angles or parallel, to release the adhesions between them and the deep parts. Grafting by Thiersch's or some other method is, however, more successful. (5) I find ultra-violet rays (three-minute doses once a week), high-frequency currents and galvanism very useful.

(b) The *contagious ulcers* are hard chancre, soft chancre, the ulcer at the seat of inoculation of glanders, and tropical ulcers due to the poisonous stings of certain insects, and the inoculation of certain tropical diseases.

(c) *Neuropathic ulcers*—e.g., perforating ulcer in tabes dorsalis.

(d) *Infiltrating* or *neoplastic ulcers* are due to the breaking down of some infiltration which has invaded the skin or subcutaneous tissue, and which can be detected in the tissue around—such as syphilis (rupial ulceration and breaking down of a gumma), lupus vulgaris, tuberculosis of the glands breaking down (strumous ulceration), epithelioma, rodent ulcer, leprosy, Bazin's disease, sporotrichosis, actinomycosis, blastomycosis, Delhi Boil, Espundia, and other nodular conditions.

The differentiation of these several varieties depends largely on the history, the associated symptoms, the distribution and character of the ulcers, and these have been given under their respective titles.

An **infiltrating, ulcerating, and scarring** *eruption in a person of young or middle age is practically either syphilis, lupus, or tuberculous ulceration. If it occurs over forty or forty-five, epithelioma and rodent ulcer enter the category.*

I. **Syphilitic Ulceration**—other than the primary chancre—is of two

kinds : (1) The breaking down of a large papular or lenticular syphilide *in* the skin gives rise to shallow irregular ulceration which may be covered with a scab which resembles the layers of an oyster shell (rupia of older authors). (2) The breaking down of a gummatous nodule which has started *beneath* the skin produces a deep punched-out ulcer. The three characteristic signs about all syphilitic ulcerations are—(1) the peripheral ring of infiltration, (2) the punched-out edge, and (3) the comparatively rapid march. The diagnosis from lupus and rodent ulceration is given in tabular form below.

II. **Lupus Vulgaris** may ulcerate, but only when near a mucous orifice, or subjected to injury and secondary infection. The nodules around are sufficiently characteristic (§ 521).

<div align="center">TABLE OF DIAGNOSIS.</div>

Syphilitic, Rupial, or Gummatous Ulceration.	Ulcerating Lupus.	Rodent Ulcer.
Anywhere.	Chiefly face.	Chiefly face.
Adult life.	Begins between ten and twenty years.	Over forty.
Progress rapid, destroying in weeks what others do in months.	Very slow.	Slow.
Sharp, clear-cut, punched-out, deep.	Edge rounded, sloping, and surrounded by apple-jelly nodules; superficial.	Edge rolled and *hard*; deep in later stages.
Discharge copious; offensive.	Scanty, inoffensive.	Scanty, and in later stages sanguineous.
KI and Hg efficacious.	No good.	No good.

NOTE.—A small piece may easily be removed for microscopic examination in any doubtful case.

III. **Tuberculous or Scrofulous Ulceration** may occasionally take its origin from tuberculous deposits in the skin, but more frequently spreads from a caseating gland or strumous bone disease. It is usually chronic. The edges of the ulcer are dark purple, and undermined, never rounded and clean-cut as in syphilitic ulceration, and there are generally evidences of the strumous diathesis, or scars from past disease of the same kind. The patients are usually children, occasionally an old person (senile struma); and see Bazin's disease (§ 522).

IV. **Rodent Ulcer** is a basal-cell carcinoma and clinically is a variety of epithelioma in which the loss of tissue is more pronounced than the new growth; it is found chiefly on the upper half of the face after forty years of age, and in the course of years will destroy the eyelids, eyeballs, and a great part of the face. It appears as a very shallow, roundish ulcer, with a flat base, scanty viscid secretion, and a very hard, " rolled " edge matted to the subjacent structures. Its progress may extend over many years, but it may at any time take on the clinical and histological characters of epithelioma in the evolution of the case.

V. **Epithelioma** is apt in course of time to undergo ulceration, but the diagnosis rests upon the characters of the initial growth, which is always found in considerable quantity around when the stage of ulceration is reached. If a small piece can be examined under the microscope the typical " cell-nest " growths of epithelioma (which are absent in rodent ulcer) can be seen.

The treatment of *neoplastic ulcers* is of four kinds : (1) Constitutional treatment

appropriate to the nature of the lesion ; (2) caustics and escharotics applied regularly and freely, so as to destroy the neoplasm around ; (3) various operative procedures—, removal, scarification, diathermy, or cauterisation ; and (4) the use of the X-rays or radium in expert hands   Excellent results have been obtained by the X rays and radium in the treatment of rodent ulcers and superficial epithelioma   Carbon dioxide snow and diathermy give beautiful results in some cases of rodent ulcer.

GROUP VII. WARTS AND EXCRESCENCES

§ 524. This group, which does not include neoplastic formations referred to in the last two groups, consists of verruca (wart), condyloma, corns, rupia, keratodermia, papilloma lineare, acanthosis nigricans, porokeratosis, and angiokeratoma

Verruca, wart, or papilloma cutis, is an excrescence consisting of thick-

FIG 162.—VERRUCA NECROGENICA on the hand of a gamekeeper aged thirty five

ened epidermis containing elongated papillæ.   Warts may occur singly, or they may be multiple.   They are most frequently met with on the hands. More rarely they occur on the head, face, or genital organs.   Warts are supposed to be due to a filter-passing virus.   They are undoubtedly in some cases contagious, and spread over the hands and other parts of the body.

Varieties.—1. *V. vulgaris* occurs on the hands, and forms a horny growth the size of a small pea. 2. *V. plana*, a flat, dark brown elevation, found on the face or back of old people. 3 *V acuminata* is moist, sessile, or pedunculated, usually quite small, but may grow to be as large as the fist, occurs chiefly on the genital organs, or where opposed surfaces are in contact, and resembles condylomata. 4. *V. seborrhoica* is an oily,

fawn-coloured, slightly elevated and rounded body, in which the papillæ are mixed with inspissated sebum. *V. necrogenica*, or "post-mortem wart," is a tuberculous infection of the skin which appears on the hands of doctors, post-mortem porters, leather-dressers, cooks, butchers, etc. It starts as a crimson, flat, indurated papule, which spreads, and sometimes becomes pustular, the pus drying and forming into a scab. A white and pinkish stellate cicatrix may be left behind as the disease progresses (Fig. 132). Warts are best dealt with by applications of glacial acetic or nitric acid, caustics, or salicylic acid plaster. Small doses of mag. sulph. t.i.d. have cured some cases. X-rays and carbonic acid snow are used in obstinate cases. Diathermy is a simple method for those who possess the apparatus. *V. necrogenica* is treated like lupus.

Syphilitic Condyloma is really a papular syphilide occurring (1) on the mucous membranes ; (2) near the junction of mucous membrane and skin ; or (3) where opposed skin surfaces are in contact. They very commonly occur at the angles of the mouth, and between the buttocks or labia. They are slightly raised discs of various sizes, covered with greyish epithelial or soddened epidermal flakes, and exuding a highly contagious fluid.

Corns are localised thickenings of the epidermis consequent on localised pressure. The side of the toe is a common position. They may be cured by painting with salicylic acid (20 per cent.) and collodion every night for a week ; soon afterwards the corn will flake off. *Soft corns* arise between the toes, due to hard corns becoming soddened with perspiration. Treatment consists in keeping them dry with dusting powder, such as zinc oxide and starch, keeping the toes separate with small pads of cotton-wool, and by relieving pressure.

Papilloma Lineare has been described under Ichthyosis, § 507.

Keratodermia may occur in (i.) syphilis. In the tertiary stages it appears as a very thickened brownish hyperkeratosis of the sole of the foot, usually associated with the thickening of the whole leg. It may also occur in the secondary stage, which is bilateral and not usually so marked in degree. (ii.) Gonorrhœa, when accompanied by severe constitutional symptoms, may show a symmetrical horny eruption on the soles of the feet. Under the horny covering are dark aloe-like nodules. (iii.) Keratodermia palmaris and plantaris (tylosis) is a family and hereditary hyperkeratosis which may have marked horny excrescences on palms and soles. In debilitated subjects secondary *syphilitic* lesions may ulcerate, with a dried blood-stained crust, which is compared to a limpet shell, and is known as *Rupia*. Similar high crusts may occur in *psoriasis*, but these have no underlying ulcer.

Acanthosis Nigricans is a rare condition characterised by progressive pigmentation of the skin, with papillary growths, terminating fatally in a few years. The colour of the skin varies from a sallow hue to bronze and dirty brown. It is generalised, but more pronounced in the flexures. The disease may occur at any age after childhood. In most of the recorded cases it has been associated with abdominal cancer, but in others no cause has been found.

Porokeratosis, a very rare disease, occurring chiefly on the backs of the hands and on the feet, is characterised by patches of atrophic skin, surrounded by a thin horny ridge or "wall" immediately inside which are seen tiny grey papules, which can be picked out. It is said to be a hyperkeratosis of the mouths of the sweat glands with destruction of glands and hair follicles. It starts in childhood and progresses slowly.

Angiokeratoma is a rare condition consisting of telangiectases, which develop into warty growths, occurring usually after chilblains, on the backs of the fingers, toes, hands, and feet. *Treatment* consists in employing warmth and electrolysis.

GROUP VIII. ATROPHIES AND SCARS

§ 525. Scars, scleroderma, and atrophy of the skin may be considered together, because they not only resemble each other clinically, but fibrosis of some of the cutaneous tissues and atrophy of others occurs in varying degrees in all three conditions. The disorders met with in this group are : I. Scars. II. Atrophoderma. III. Scleroderma. IV. Keloid and its congeners Rhinoscleroma, Kraurosis Vulvæ, and Ainhum.

I. **Scars** may result from burns, wounds, or infiltrating or suppurating eruption in which there has been a loss of substance. If much deformity or loss of mobility results, plastic operations are called for ; but it is wonderful how much can be done in young patients by means of persevering massage with oleaginous substances, and especially, in my experience, cod-liver oil. Scars are liable occasionally to be affected by keloid (see below).

II. **Atrophoderma** (Atrophy of the Skin) occurs as : (*a*) Atrophy of the entire cutaneous covering is common in old age ; (*b*) Linæ albicantes is a term applied to the atrophic streaks found on the abdomen and breasts after pregnancy, over the hips and other parts when the patient has been getting rapidly stouter. Linear atrophoderma (Striæ atrophicæ) has been noted after fevers, especially pneumonia and enteric. They are distinct from the linear albicantes, which follow distension of the skin. The author and others have also observed cases which could be traced to a neuropathic cause. (*c*) *Unilateral atrophy* of the skin is met with in the condition known as Hemiatrophy Facialis (§ 656), which is of nerve origin.

III. **Scleroderma**, or fibrous thickening of the skin, is met with in three clinical forms, all of which are more or less rare : (*a*) Localised (or morphœa) ; (*b*) diffuse ; (*c*) S. neonatorum.

(*a*) LOCALISED SCLERODERMA (Synonyms : Morphœa of Erasmus Wilson) is a disease consisting of one or more localised ivory patches of sclerosed skin with, in the earlier stages, a congested lilac border. The patch may be atrophic and pigmented. There are few or no subjective sensations, but the tactile sensation is diminished. Some cases undergo spontaneous resolution in course of years. The favourite situations are the face, neck, and beneath the breast. There is a tendency to symmetry. It is thought that the shape and distribution of some patches corresponds with the distribution of a nerve, the supra-orbital being a common site, but it seems more probable to the author that the lesions are associated in some way with Head's sensory and visceral areas. Females are more prone to be affected in the proportion of three to one, and the disease appears mostly in the first half of life. Beyond the disfigurement and contraction the patient suffers but little inconvenience from this variety of the malady.

(*b*) DIFFUSE SCLERODERMA is a somewhat different affection to the foregoing, and consists of a parchment-like thickening and contraction of the skin. When it starts and predominates in the extremities, it is called sclerodactyly. It progressively increases until the parts become completely hidebound and immobile. The face in such cases wears a smooth, expressionless aspect. This disease is one of much gravity. By degrees fissures and ulcers form, and some of the fingers may become gangrenous, and death from some intercurrent malady occurs. Many degrees of severity are met with, and in some the condition only produces a constant liability to cold and to various superadded skin lesions.

(*c*) SCLERODERMA (Synonym : Sclerema) NEONATORUM is a different disease. It appears congenitally, and is generally fatal in the course of a few weeks. The affected skin is bound down to the parts beneath in livid, tense, shining patches, which tend to become universal.

The *Treatment* of the three conditions is not satisfactory. Hot air baths and local massage may be recommended. Several slighter degrees of generalised scleroderma

under my care improved under thyroid. The constant current, hot air and light baths, and fibrolysin have done good in the localised form. Electrolysis does good in morphœa.

IV. **Keloid** consists of a fibromatous deposit in the skin occurring primarily in unaffected skin, or secondarily, in old cicatrices. The lesion appears as a small firm nodule, of a crimson or pinkish colour, which slowly enlarges by means of tentacle-like processes. At first it is raised above the skin level. Keloid can be distinguished from a hypertrophic scar only by microscopic examination. If excised, keloids immediately recur, and in that sense they are malignant. The negative pole of a mild constant current gives favourable results. Fibrolysin injections and X-rays in pastille doses are useful.

Acne Keloid (Synonyms: Dermatitis Papillaris Capillitii, Sycosis Capillitii) is a rare disease which occurs on the nape of the neck, a slow pustular affection, resulting in keloid formation.

Rhinoscleroma is a chronic inflammatory affection characterised by the development of hard, circumscribed, nodular growths in the skin and mucous membrane, most commonly of the nose and naso-pharynx, due, it is believed, to inoculation with a specific bacillus. The bones and cartilage may be involved.

Kraurosis Vulvæ begins with red patches, followed by atrophy and contraction of the mucous membrane of the external genitals and adjacent perineum of women. The tissues are atrophied with thickened patches.

Leukoplakia Vulvæ is a chronic condition, with red patches round the urethra and entrance to the vagina. Cracks and bleeding fissures may follow. As it tends to become malignant, early excision of the involved areas is advisable.

Ainhum consists of a slow strangulation and amputation of one or more toes by the growth of a constricting band of hypertrophied skin. It is not seen in this country.

GROUP IX. PIGMENTARY AND VASCULAR ALTERATIONS

§ 526. Alterations of colour depend mainly upon the condition of the vessels and the amount of pigment in the skin. Pigmentation of the buccal mucous membranes is seen chiefly in Addison's disease and Pernicious Anæmia. A *diminution* of pigment is not frequent, and occurs only in two conditions: (1) ALBINISM, a congenital condition in which there is deficient pigment in the skin and its appendages, and in the iris and choroid; and (2) LEUCODERMA (Synonym: Vitiligo), a condition in which there is an absence of normal pigment in areas which are surrounded by darker-coloured skin. The transition from the pale to the dark area is abrupt, and it is the dark-coloured concave margins which attract the notice of the patient. It may be congenital or acquired and is probably neurotrophic in its origin.

a. A *localised* increase of pigment or alteration in colour occurs in:

I. Chloasma.	VII. Xeroderma pigmentosa.
II. Lentigo.	VIII. Xanthoma.
III. Pityriasis versicolor.	IX. Morphœa alba and nigra.
IV. Pigmentary and vascular moles.	X. Ochronosis.
V. Purpura.	XI. Leprosy.
VI. Urticaria pigmentosa.	XII. Von Recklinghausen's disease.

b. A *generalised* increase of pigment occurs in (1) arsenical and silver pigmentation; (2) Addison's disease; (3) abdominal cancer; (4) cardio-vascular disease; (5) bronzed

diabetes; (6) constipation; (7) melanotic sarcoma; and (8) acanthosis nigricans; but in these the pigmentation is subordinate to other symptoms.

I. **Chloasma** occurs in single or multiple patches of diffuse discoloration on various parts of the body, varying in shade from a light yellow to a deep brown. Several varieties may be referred to: 1. *Chloasma symptomatica* is met with most frequently in pregnancy or uterine disease, and its most usual position is on the face and round the nipples. 2. *Chloasma cachecticorum* occurs in association with malaria, cancer, senile atrophy, rheumatoid arthritis, abdominal tubercle, or cancer, and exophthalmic goitre. Pigmentary syphilide may take this form, and is usually seen on the neck. 3. *Chloasma traumatica* is the pigmentation beneath the garters, or around the waist in tight-lacing women, in pediculosis or scratching, and after sinapisms, blisters, etc. In this category may be included the pigmentation which follows chronic eczema, scratching, syphilis, lichen planus, psoriasis, or any other long-continued afflux in the skin capillaries. 4. *Chloasma caloricum* is the pigmentation due to sun and wind, or to heat, as on the shins of women who sit over the fire. It also follows exposure to X-rays.

II. **Lentigo** (Synonyms: Freckles, E p h e l i d e s).— Freckles are multiple, circum-scribed pigment spots on the portions of the body exposed to light. Hyd. perchlor. ¼ per cent. in alcohol may be tried cautiously; paint on twice daily till scaling ensues. In advanced age freckles may occur anywhere, and are apt to become malignant.

III. **Tinea V e r s i c o l o r** (Synonym : Pityriasis Versi-color) is a vegetable parasitic affection of the skin, which appears as variously sized, irregularly shaped, dry, highly furfuraceous patches, yellow-ish-brown in colour, found generally upon the trunk, and especially in the hollow of the breast bone, due to a specific parasitic fungus, the micro-sporon furfur (Fig. 133).

FIG. 133.—MICROSPORON FURFUR, the fungus of PITYRIASIS VERSICOLOR, × about 50.--Shows the branching irregular mycelium and the constellations of spores. Stained by Gram's method.

IV. **Pigmented and Vascular Moles** are distinguished from other pigmented conditions by their being congenital, and being more or less raised. Nævus spilus is a smooth discoloured spot of otherwise healthy skin. Nævus verrucosus is rough, and sometimes bristling with hairs (nævus pilosus). *Vascular moles* are purple spots of increased vascularity, usually a little raised above the surface, varying widely in size and thickness. They are sometimes stationary, but more often gradually increase in size. *Telangiectasis* is a localised dilatation of the vessels of the skin, and is a frequent accompaniment of acne rosacea. Small spots are met with independently of any skin affection on the face and various parts of the body in healthy persons, particularly as life advances, or when the peripheral circulation is feeble. *Port-wine mark* is a venous and capillary dilatation over an area of skin. *Multiple Telangiectases* occur as a familial and hereditary disease which is transmitted by male or female to both sexes. Multiple small nævi occur, and those on the nasal or other mucous membranes are very apt to bleed and cause serious, even fatal disability. Sometimes the nævi occur only on the nasal mucous membrane, giving rise to repeated epistaxis.
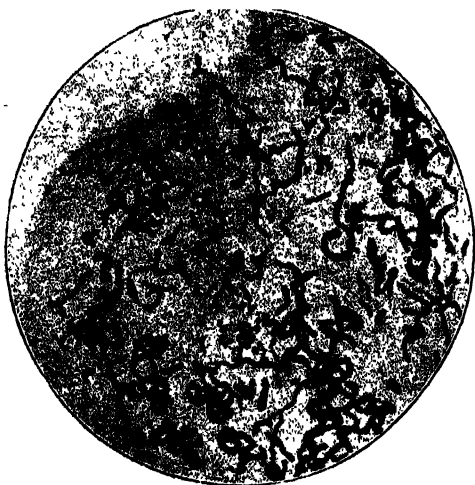
Treatment.—Leucoderma, lentigo, and chloasma are best treated with strong mercurial lotions (¼ to 1 per cent.), but they are difficult to cure. The general health requires treatment in leucoderma and chloasma. For pityriasis versicolor thorough cleansing with a hard brush and soap is essential, together with ung. sulph. or a lotion of sod. hyposulph. (1 drachm to the ounce). The underclothing must be disinfected. Electrolysis, escharotics, or excision give good results in pigmented and vascular nævi ; for the latter carbon dioxide snow and diathermy are now widely employed.

§ 527. V. **Purpura** consists of dark, abrupt-edged purple spots due to extravasations of blood into the skin. The patch does not fade on pressure. It is sometimes accompanied by similar extravasations into the mucous membranes and internal organs, and hæmorrhages from the mucous surfaces. The constitutional symptoms vary considerably, and may be absent. Pyrexia, usually slight, occurs in about half the cases.

The *Causes* of purpura are but little understood, but may be grouped under three headings : 1. *Local* and *mechanical* causes, such as *heart disease* and *old age.* The most frequent illustration of this is met with in eczema of the leg (indeed, most eruptions on the legs of old people), which assumes a purpuric character on account of the hypostasis of the blood. Mitral disease is associated not infrequently with purpuric eruptions on the legs. 2. Purpura is associated with certain *nervous* conditions, such as cerebro-spinal meningitis, tabes, myelitis, hysteria, and, according to some, neuralgia. 3. *Various infections of the blood* produce purpura in (i.) *fevers*, especially typhus (typhus has been called purpura contagiosa), pneumonia, and cerebro spinal fever, in which a purpuric eruption is frequent ; measles sometimes ; malignant endocarditis, and pyæmia occasionally ; the initial stages of small pox, and in the *malignant type* of that and most of the other acute specific fevers ; the rheumatic poison, pyogenic organisms ; (ii) perverted metabolism, certain articles of diet inducing the condition in some people ; (iii.) certain *drugs* occasionally, especially mercury and iodide, quinine, copaiba, belladonna, ergot, salicylic acid, and the mineral salts ; (iv.) *autotoxic* conditions, such as Bright's disease, hepatic disease, including cirrhosis, acute atrophy, carcinoma, and any aggravated jaundice ; bad food also seems in some cases to account for the onset of purpura ; and (v.) *constitutional* diseases, such as leukæmia, scurvy, lymphadenoma, and great debility from any cause.

Four special *varieties* of purpura are recognised. *Purpura Simplex* (morbus maculosus of Werlhof) is the name given to a mild attack of purpura for which no cause can be assigned. It is usually met with in young persons, and runs a benign course. *P. Hæmorrhagica* is a severer variety, which may start as P. Simplex, or independently. The spots are larger, and sometimes raised or œdematous (P. urticans). They may be capped with bullæ. Hæmorrhage occurs from the mucous membranes, and pyrexia is more marked. This form of purpura has received more attention of recent years owing to its analogy with anaphylactic phenomena, and its association and alternation with angio-neurotic œdema. The individual may have signs of both conditions in different parts of the body. Hæmorrhage may occur into the alimentary canal, kidney, eyes and brain. Vomiting and colic may occur at the same time as, or alternate with, periods of outbreak of skin lesions. These conditions have been called `P. Rheumatica* (Peliosis Rheumatica, Schönlein's disease), and *Henoch's Purpura*, described in § 466. Purpura, angio-neurotic œdema and erythema are related ; the first is due to an exudation of all the constituents of the blood, the second to an exudation of the serum ; the third to both combined. Consequently it is not surprising to find that many intervening stages are met with.

The *Diagnosis* of purpura is easy, but difficulty lies in ascertaining its cause. The diagnosis from *scurvy* has been given (§ 440). Consider the previous history, and thoroughly examine every organ and the blood. In *hæmophilia* the blood clots slowly and the clot contracts : in purpura the clot does not contract. (*B.M.J.,* March 30, 1912.)

The *Prognosis* is extremely grave when associated with the specific fevers, or with a high temperature. P. simplex usually results in recovery in a few weeks. P. rheu-

matica is rarely fatal, though it may last for months or years, and may recur; its complications may be serious.

Treatment.—Arsenic, iron, ergot, and ol. terebinth deserve trial. Calcium chloride and adrenalin are useful. Twenty q.c. blood serum, taken from a healthy individual, and injected intramuscularly, has given good result in severe cases.

VI. **Urticaria Pigmentosa** (described by Nettleship in 1869, and so named subsequently by Sangster) is a chronic condition with spots of brown pigmentation; when rubbed, these develop wheals. The disease starts in early childhood; it may cease spontaneously about puberty, but it more frequently lasts for many years. The *Treatment* is like that of urticaria.

VII. **Xeroderma Pigmentosa** is a rare disease of a chronic progressive character, starting in early childhood, often in members of the same family, and marked by small dark freckles, with subsequent atrophy and contraction of the skin, and the occurrence of telangiectases. There is also a distinct tendency to a malignant new growth, both in the skin and the internal organs in the shape of a malignant sarcomatous or cancerous infiltration. The distribution is universal, and the contraction gives rise to eversion of the eyelids and other orifices. It usually terminates in death before the age of twenty-two. The only treatment is protection from light.

VIII. **Xanthoma** (Synonym : **Xanthelasma**) is a rare condition most commonly occurring in cases of diabetes and chronic jaundice. It consists of yellowish flattened patches which usually occur on the eyelids ; or as nodular deposits on limbs or trunk, varying in size from a millet-seed to a bean, or larger (cp. p. 724). It is associated with excess of cholesterol in the blood. Caustics and electrolysis remove the lesions.

IX. **Morphœa Nigra** and **Morphœa Alba** are names employed by Erasmus Wilson for the disease which we now describe as localised scleroderma (§ 525), when attended by excess or deficiency of pigment.

X. **Ochronosis** is a rare disease characterised by blackening of the cartilages and ligaments and fibrous tissue beneath the skin. The sclerotics and extensive areas of the skin may show black pigmented patches. There is sometimes arthritis, and alkaptonuria (§ 313) is often associated ; rarely, there is carboluria, due to frequent use of carbolic acid.

XI. **Leprosy** (§ 522).—Patches of pigment and white spots may occur in the early stage of anæsthetic leprosy, and dark spots occur, especially on the face, in the early stage of nodular leprosy.

GROUP X. DISORDERS OF THE SWEAT

§ 528. Four disorders of sweat are met with ; Anidrosis, hyperidrosis, bromidrosis, and chromidrosis.

I. **Anidrosis** is not common apart from the conditions mentioned in Group VIII.

II. **Hyperidrosis** is the term applied to an excessive secretion of the perspiration, and may be general or localised. When general it may be due to a lowered neurovascular tone, excitement, corpulency, or the use of stimulating foods and drinks. It also occurs in fevers at the crisis, in ague, acute rheumatism, chronic tuberculosis. The *localised* form affects most often the feet, axillæ, and palms. The sweat mixed with sebaceous secretion decomposes on the clothing, and gives rise to a pungent and disagreeable odour. The feet are apt to become tender, and eczema may supervene.

The *Treatment* of hyperidrosis consists of the application locally of a mixture of tr. belladonna and water equal parts, or sponging with vinegar and water ; and internally hypodermics of atropin, or the administration of arsenic and the mineral acids or tonics. *Local* hyperidrosis, especially of the feet, is troublesome to get rid of. The stockings should be changed several times a day, and put into a saturated solution of boracic acid before being used again. Dusting powders relieve the slighter forms. Applications of tannic acid, chromic acid, salicylic acid, and of diachylon plaster should be tried in graver cases. In severe hyperidrosis of the axillæ I have had good results with X-rays, but ten years later telangiectases supervened.

III. **Bromidrosis** is the term applied when the perspiration has an offensive odour. Mere excess of perspiration in the feet or axillæ may render the person disagreeable to his companions. The subject is therefore interwoven with hyperidrosis, to which reference should be made for the treatment.

IV. In **Chromidrosis** the perspiration is coloured.

GROUP XI. DISEASES OF THE SCALP AND HAIR

§ 529. The diseases special to the scalp and hair are :

I. Ringworm.	VII. Hirsuties.
II. Favus.	VIII. Trichoptylosis.
III. Alopecia.	IX. Trichorrexis Nodosa.
IV. Pityriasis and Seborrhœa.	X. Leptothrix.
V. Canities.	XI. Tinea Imbricata.
VI. Pediculi Capitis.	

I. **Ringworm** (Synonyms : Trichophytosis Capitis, Tinea Tonsurans) may be caused by the small-spored fungus, microsporon Audouïni, and by the large-spored fungi, the Trichophytons endothrix and ecto-endothrix (see Figs. 134 and 135). The clinical appearance varies with the form of
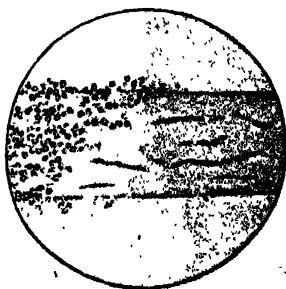

FIG. 134.—SMALL-SPORED RING-WORM (Microsporon Audouïni) under a ⅓-inch objective.—Spores forming a thickset sheath outside hair; mycelium within the hair branching and irregular, not parallel.
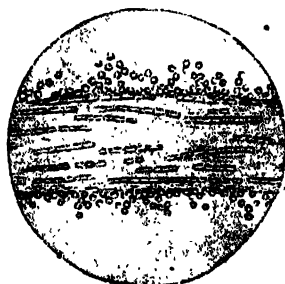
FIG. 135.—LARGE-SPORED RING-WORM (Megalosporon). — Mycellum within the hair parallel and breaking into equal segments with truncated ends.

infection. About 90 per cent. of the cases occurring in England are due to the microsporon, but this variety is somewhat rare abroad. It starts as an insignificant, semi-bald, pink patch, usually overlooked, and when first seen by the physician is a white, powdery, circular patch on the scalp of children, with *broken hairs*. The spot varies from the size of a three-penny piece to one denuding half the scalp. In the variety due to the endothrix there may be few or no scales on the patch ; broken hairs are found at its margin, and on the patch itself, which may be quite bald, there are often black dots due to the hairs being broken off level with the scalp. When the ecto-endothrix affects the scalp, kerion is common —a condition with deep-seated boggy suppuration.

Diagnosis.—The broken hair stumps are quite characteristic. Dabbing chloroform over the part will reveal the diseased hairs, which then look

whitened like hoar frost. The diagnosis should be clinched by placing the hair on a slide with a drop of liquor potassæ, and examining under the microscope. The varieties of the fungus, however, can only be distinguished after staining by Gram's method (§ 667), and by their culture characteristics. A bald form of ringworm occasionally occurs resembling *alopecia areata*, but some broken stumps of hairs can be found at the margins of the patches.

Etiology.—The disease is rare in children over fourteen, but common under ten. As a rule the small-spored form spreads rapidly from child' to child in schools and families ; adults are rarely affected. Both small- and large-spored ringworm can be communicated from animals—dogs, cats, cattle, horses and birds. In the tropics other fungi are found.

Prognosis.—The disease lasts an indefinite time, but tends to disappear spontaneously about puberty. Much depends upon the stage at which the disease is first seen, the diligence of the treatment, and the variety of spore present, the small spore being much more intractable. Fair-haired children are more difficult to cure. The average duration is two or three years. Varieties with kerion usually run a shorter course.

Treatment.—Ringworm is a most difficult malady to cure, and great perseverance is required. The head should be shaved every ten days, and a linen cap worn which can be renewed every two or three days, the old one being burned. When the disease is not widespread, I have found an efficacious lotion consisting of picric acid, gr. vii. (0·4), camphor and rectified spirit, aa ℥ ss. (16) (A.S.). Patches seen very early may be aborted with strong parasiticides—*e.g.*, pure carbolic acid, iodine, or croton-oil. Every day (1) cleanse and remove all débris with A.C.E. mixture ; (2) apply parasiticides. Air and water should, as far as possible, be excluded. Amongst the numerous remedies may be mentioned salicylic acid (10 grains to the ounce of collodion), chrysarobin, carbolic, mercury, and oil of cade in varying strengths ; and it is well to change the remedies at times. It is often very difficult to decide when a child is free from infection. My own method is to leave the case which appears cured without treatment and untouched for ten days. If at the end of that time the surface is free from scaliness, the hairs are growing normally, and the microscope gives negative results, I recommend further treatment for a week, and then, after a second interval of ten days, if the same tests answer, I pronounce the case as probably cured. When the services of a dermatologist skilled in the use of the X-rays can be obtained, the quickest and best method of curing ringworm is to epilate the hair by the Sabouraud method of X-ray administration.

II. **Favus** occurs on the head and the body. It is rare in England, but commoner in Scotland. It forms such characteristic irregular yellow crusts, with yellow, cup-shaped tops, and is accompanied by such a mousy smell, that the diagnosis is not difficult. The microscope reveals the spores and the mycelium of the achorion Schonleinii (Fig. 136). It develops slowly, is accompanied by itching, and leaves atrophic scars. It is less contagious than ringworm, but more intractable. It may

spread to the body. The treatment is the same as in that disease, but epilation is more necessary.

III. Alopecia (Baldness) may be congenital or acquired, partial or complete, diffuse or in patches. The acquired condition may arise from (1) *premature senility*, which usually begins on the vertex; (2) *general malnutrition*, when the hairfall is diffuse, as in tuberculosis, acute fevers, anæmia, and nervous exhaustion; (3) *syphilis*, in which the baldness is disseminated or patchy in the early stages, or localised in the later stages of the disease (being then due to syphilitic lesions of the skin); (4) *favus and ringworm*, which affect the hairs and lead usually only to temporary and localised baldness; (5) *lupus erythematosus*, in which the bald patches are permanent; (6) impetigo, eczema, X-rays, etc., with temporary loss of hair; (7) *seborrhœa capitis*, which is probably the commonest cause of baldness in men; (8) *pityriasis capitis*, a common cause, especially in women; (9) two forms of cicatricial alopecia are met with rarely—*pseudopelade* of Brocq, and *folliculitis decalvans*.
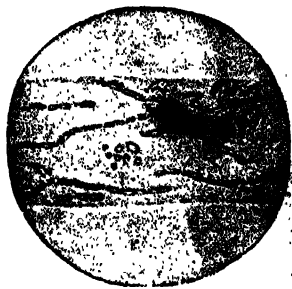

Fig. 136.—FAVUS FUNGUS (tinea favosa or achorion Schönleinii) magnified about 300.—Groups of irregular-sized spores within the hair; mycelium irregular in thickness and arrangement.

Alopecia Areata is a special form of baldness occurring in circular patches which are smooth and white. Each patch slowly increases peripherally, and at the margin short diseased hairs may be seen, which have so characteristic an appearance as to enable us at once to identify the disease. The free end is of normal thickness, but presents a ragged fracture where the hair has been broken off; from this point the shaft gradually becomes thinner towards the root, which is extremely atrophied. Thus it somewhat resembles a note of exclamation (!). Any part of the body may be affected. The disease runs a very protracted course, lasting, if untreated, for years. In course of time a few downy hairs begin to grow, white at first, but gradually becoming coloured. It is undecided whether the disease is of parasitic or neurotic origin.

In the *Treatment* of baldness attend to the general health, and apply local remedies in order to stimulate the vascularity of the part by means of lotions or ointments, commencing with ammonia, turpentine, and cantharides. If seborrhœa or pityriasis is present, they must be corrected. The treatment of the bald scars left by lupus erythematosus and other scarring eruptions of the scalp is hopeless, for the hair-bulbs are destroyed. Jacquet found that alopecia areata was often due to reflex irritation from the teeth and naso-pharynx, whilst Barber and Leslie Roberts found certain cases recovered when chronic infective foci in the tonsils and sinuses were dealt with. In the average case of alopecia areata the patient should be assured that a prolonged course of treatment is necessary. Galvanism, high-frequency currents, massage and especially ultra-violet light and

X-ray are useful. In alopecia due to the microbacillus of Sabouraud, a form which is associated with excessive oiliness, vaccines yield good results in young subjects.[1]

IV. Seborrhœa Capitis Sicca is the name usually given (wrongly) to cases of Pityriasis Sicca (dandruff) occurring in localised patches or generalised over the scalp. It is due to the bottle bacillus. It may be accompanied by a degree of inflammation due to the presence of staphylococcus griseus (Sabouraud). It is diagnosed from eczema by the greater inflammation and exudation in the latter ; and from psoriasis by the larger number and size of scales in psoriasis. An abnormally greasy scalp is found with seborrhœa oleosa, which is due to, or at least associated with, the microbacillus of Sabouraud. Both pityriasis and seborrhœa oleosa may lead to diffuse hairfall and alopecia.

The *Treatment* consists in washing the head once or twice a week with equal parts of soft soap and spirits of wine, and in rubbing in every night a lotion or ointment containing mercury, tar, or sulphur.

V. Canities, or whiteness of the hair, is, as the name implies, most usually an evidence of advancing years, or of overwork, sudden or prolonged grief, defective general health, or neuralgia. The *Treatment* is unsatisfactory apart from the improvement of the general health by tonics. The head should be examined for seborrhœa, pityriasis, or any other local disease, which, though it does not cause, may expedite the loss of colour.

VI. Pediculosis Capitis presents the following features : (1) The pediculi (Fig. 137) ; (2) white specks on the hairs (the eggs or " nits "), which cannot be pulled off, by which they are distinguished from dandruff (Fig. 138) ; and (3) irritation and inflammation. If the condition is untreated, there results pustulation with formation of thick crusts, matting of the hair, and enlargement of the occipital glands. Kill the living parasite and also the nits. This may be done by soaking the hair with methylated spirit or kerosene for one or two nights, or bathing with 1 in 20 carbolic, then combing the hair thoroughly with a small-toothed comb. Ungt. hyd. amm. may be rubbed in. The nits may be dissolved by washing the hair with strong vinegar.

VII. Hypertrichosis (Synonym : Hirsuties) is a growth of hair either abnormal in amount or in position, and the most troublesome of these is the moustache or beard found on the faces of some women. *Treatment* consists of the removal either by the razor, diathermy or electrolysis. Depilatory pastes do not prevent regrowth. The X-rays are efficacious, but are followed by telangiectasis many years later.

VIII. Trichoptylosis, or splitting of the ends of the hairs, is met with sometimes in women. It often occurs in association with deranged general health.

IX. Trichorrexis Nodosa is a very rare disease in which a series of spindle-shaped swellings appear upon the hair, which break transversely, and leave a brush-like extremity.

X. Leptothrix (Synonyms : Mycosis Axillaris, Trichomycosis Nodosa) is a disease affecting the hairs of the axillæ and scrotum. The hairs are dry and knotty, due to adherent small concretions, which may affect the whole length of the hair, but not the follicle, or may occur as separate nodules on a hair. Bacilli are found in these concretions, and the hair may be split longitudinally (Fig. 139).

XI. Tinea Imbricata is a contagious disorder of the tropics due to fungus. Its configuration has a watered silk appearance.

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[1] "Treatment of Seborrhœa," by Agnes Savill, *The Practitioner*, 1911 and 1913.

§ 580. General Remarks on the Treatment of Skin Diseases.—There are three principles upon which we must depend for success : (1) If we except mercury and iodides in syphilitic and other granulomata, and perhaps arsenic in lichen planus, and some cases of dermatitis herpetiformis, there are no such things as specific remedies in skin diseases. It is therefore not sufficient for purposes of treatment to diagnose a case as eczema, psoriasis, lupus, etc. First of all, to consider *local* treatment, we must recognise *the stage of the disease* and the *precise pathological process* before us. An ointment which would cure a chronic eczema would greatly aggravate an acute weeping one. It follows, therefore, that it depends not so much upon the name which we decide to give to an eruption, as upon the amount of congestion, swelling, scaling, thickening, discharge, itching, etc., which is present. We have, in a word, to treat the symptoms, the sum of which constitutes the disease at that particular moment. This requires considerable experience, and herein lies the justification of the speciality of dermatology. (2) The *method* of application of a remedy is of as much importance as the composition of it. (3) The *idiosyncrasy* of a patient and the susceptibility of his skin to various remedies must always be remembered. This is especially true of the face ; what benefits one person's skin irritates another's.
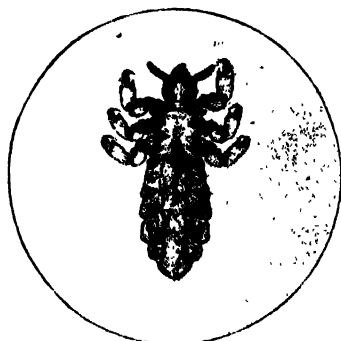

FIG. 137.—PEDICULUS CAPITIS × 10.—It differs from the pediculus corporis only in being shorter, and in its thorax and abdomen being more nearly equal in size (see p. 699).
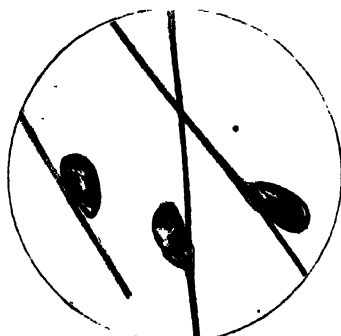

FIG. 138.—NITS (eggs) of PEDICULUS CAPITIS on hairs, slightly magnified.

It is convenient to divide external remedies into three groups : the first being indicated for acute, congestive, or moist conditions, the second for dry, scaly, hard, and chronic conditions, and the third group (caustics) having a corrosive action for the removal of diseased structures.

(a) SEDATIVES and ASTRINGENTS are used chiefly in acute conditions to reduce hyperæmia, to check exudation, and to allay pain. Enumerated in order of increasing strength the most important are zinc, lead, bismuth, mercurous, and, in a less degree, mercuric and silver salts. Zinc and bismuth are inert on the unbroken skin. These remedies are generally applied in the form of ointments, as a powder, or, still better, suspended *in lotions* for acute eczema and similar moist conditions. Zinc oleate is also a bland preparation, and zinc gelatine may be used when there is little discharge. Ung. diachyli is also soothing. Mercurial preparations are more efficacious in chronic conditions. The perchloride is irritating, and if used strong comes under the heading of caustics. Vegetable astringents are not much used, though the glycerine of tannic acid may be used for congestive conditions around the mucous orifices.

(b) STIMULATING APPLICATIONS, ANTISEPTICS, TARS, and AROMATICS are useful to stimulate chronic conditions to more healthy action. They include wood tar or creosote, and coal tar or creolin, carbolic acid, resorcin, thymol, β-naphthol, benzoic and salicylic acids—salicylic acid especially being a very useful preparation to reduce

hyperplasia of the epidermis—ichthyol, sulphur, and chrysarobin. The earlier named of these are less irritating than the later ones; indeed, weak preparations of tar may act as a sedative.

(c) CAUSTICS have a definite corrosive action upon the skin. Thus liquor potassæ may be used to remove the superfluous epidermal scales of psoriasis prior to the application of an ointment or plaster. Others are mercuric chloride, acid nitrate of mercury, nitric acid, pure carbolic acid, etc. Soft soap is the mildest of such preparations, and may be usefully added to an ointment for the treatment of inveterate, psoriasis.

(d) PROTECTIVE MEASURES.—The old-fashioned paste—i.e., an ointment made up with a large propor-tion of some powder (e.g., F. 75)—comes under this heading. Pastes are applied by laying on in a thick layer. One of the most useful forms of protec-tion is ZINC GELATINE. Painted on to the dis-eased skin this fulfils four indications: (i.) Gentle compression and support, as in varicose eczema or hypostatic congestion; (ii.) protec-tion from the action of the air or friction while allowing natural evapo-ration and healthy action to go on be-neath; (iii.) it allays itching; (iv.) is an excellent dressing and means of applying remedial agents con-stantly. Another protective agent is the PLASTER MULL introduced by Unna (Ham-burg), which consists of reagents mixed with gutta-percha and some basis, and spread upon a piece of muslin.

FIG. 189.—LEPTOTHRIX, slightly magnified.

(e) Treatment by X-rays, diathermy, high-frequency current, ultra-violet rays, and carbonic acid snow requires special training in the use of the apparatus, and experi-ence alone can decide the dosage suitable.

As regards *general* treatment, it must be remembered that a skin disease is usually only an external expression of internal disorder, and comparatively rarely a reaction to an external irritant. Hence certain forms clear up when a septic focus, constipation or other cause of toxæmia is removed. This is not necessarily the result, however. When the individual's threshold of resistance is lowered, or he has (in other words) become sensitised to a poison, the removal of one septic focus may give only temporary relief, because another is soon formed. The health as a whole must be improved, and all forms of treatment here have their sphere, from endocrine preparations, diet and vaccines to mountain air, heliotherapy and psychotherapy.
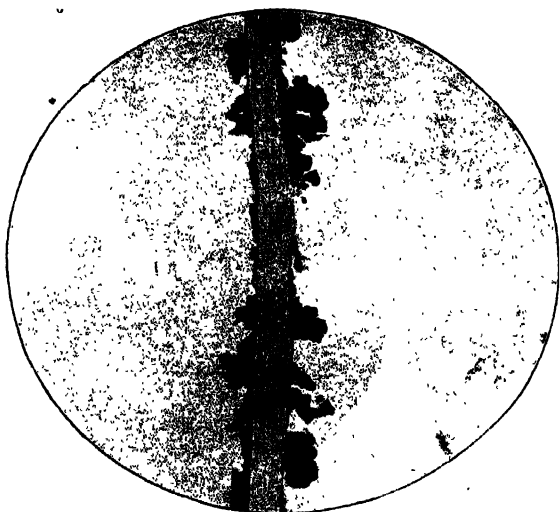
CHAPTER XIX

THE NERVOUS SYSTEM [1]

Our first duty, when dealing with a widespread structure like the nervous system, is to *localise* the seat of mischief : Is it, for instance, in the brain, cord, nerves, or autonomic system ? This accomplished, the second stage of the process, the diagnosis of the *nature of the lesion*, is not generally difficult, for the position it occupies, the age of the patient, his history, and the mode of onset of the illness will generally afford us a fairly certain basis for decision. A convenient method of examining a case of nervous disease is given below.

ANATOMY AND PHYSIOLOGY

It follows from what has just been said as to the necessity of identifying the *locality* of a nervous disease before we can diagnose its *nature*, that an accurate knowledge of the anatomy and physiology of the nervous system is essential. Anatomically the nervous system consists of Encephalon (Cerebrum, Cerebellum, Pons Varolii, and Medulla), Spinal Cord, Peripheral Nerves, and Autonomic System. But such a division is artificial, because these various parts are brought into relation with one another by an endless series of intercommunications.

§ 531. **The Neuron.**—Histologically, physiologically, and pathologically the nervous system consists of a collection of cells which with their processes are called *neurons*. The nerve " cells " are called the *neuron-bodies*, and the shorter cell processes are sometimes spoken of as *dendrons*, their ultimate branching processes being called *dendrites*. One of the processes is longer and larger than the others, and often receives a special covering in the shape of a medullary sheath after leaving the neuron-body or " cell " ; this process is called the axis-cylinder process or *neuraxon*, and constitutes a nerve-fibre. The neuraxon (except in the case of the sensory nerve-fibres) is efferent, while the dendrons are afferent in function. The neuron-bodies or cells are situated entirely in the *grey matter* of the brain and cord, or in the various ganglia, such as those of the posterior roots and autonomic system. The neuraxons in passing to their destinations are often of considerable length ; they pass through and constitute the fibres or *white matter* in the brain and cord, where they form the nerve fibres, and finally traverse the *nerve trunks* to their destination in the tissues. The neurons of the central nervous system are supported in connective tissue known as neuroglia.

Nerve-cells at one time were thought to be connected with each other by means of their branching processes, but it is now known that the extremities of the processes

are free, the intervals between the adjacent terminals of separate neurons being known as *synapses*.

Fig. 140 represents a peripheral sensory neuron or protoneuron. The neuron body in the case of the spinal nerves is situated in the posterior root-ganglion, and in corresponding ganglionic swellings in the case of the 5th (Gasserion ganglion), 7th (geniculate ganglion), 8th (cochlear nerve spiral ganglion, vestibular nerve spiral ganglion), 9th and 10th cranial nerves. Note that the nerve-cell has a single process (unipolar) which after making several coils receives a medullary sheath and neurilemma. While still within the ganglion, it divides into a peripheral and a central branch. The former constitutes a sensory nerve-fibre ; the latter enters the central nervous system as an exogenous fibre, losing, or all but losing, its neurilemma, and divides into a larger ascending and a shorter descending branch, each of which gives off at right angles, or approximately so, a number of collaterals whereby the protoneuron forms synapses with other neurons.

The student should study Fig. 141 carefully, as it epitomises much useful information. It represents the cranial and spinal protoneurons. In the lower part of the lower figure, the ascending and descending branches of a unipolar spinal protoneuron are shown to be brought into relation with the motor neurons of the anterior horns by means of an "intercalary" neuron.

Note that the acoustic and the vestibular protoneurons (8th nerve) are bipolar. This is the case with all the
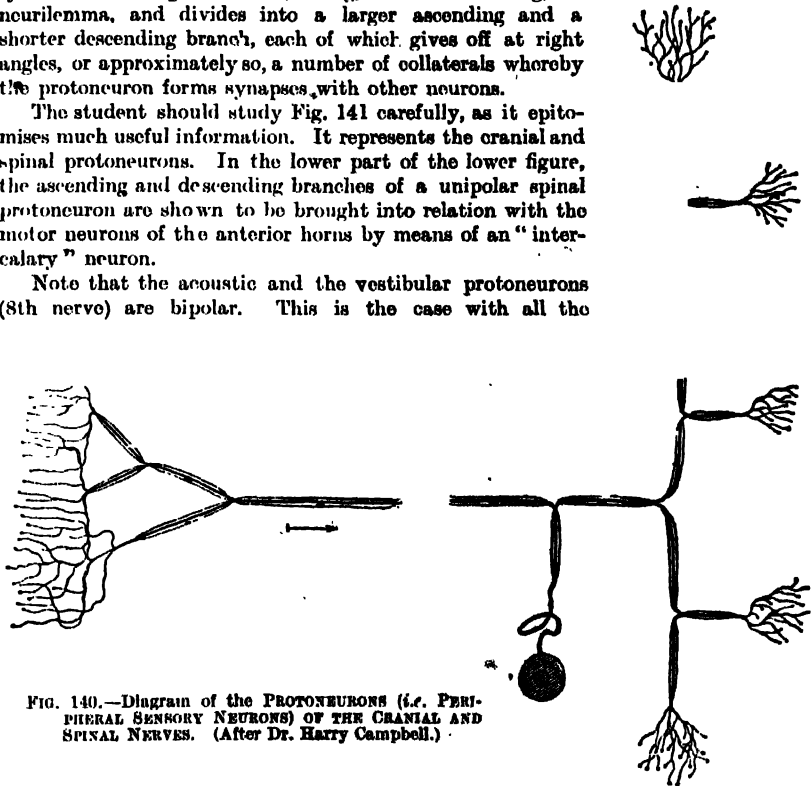
protoneurons of the lowest vertebrates. The olfactory protoneurons are still more primitive in that, besides being bipolar, they are wholly located in the periphery, *i.e.*, in the Schneiderian membrane. The like is true of the visual protoneurons, which are located in the retina. The central processes of the 7th, 9th and 10th are seen to pass to the dorsal part of the brain-stem, where by their bifurcation they form tracts which lie in the central portion of the solitary tract.

The peripheral *efferent autonomic strand* to involuntary muscle- and gland-tissue is represented in the upper portion of the lower figure. It is seen to consist of preganglionic fibres passing from the grey matter of the spinal cord to a sympathetic ganglion, and of post-ganglionic fibres arising in the latter. These travel either in a cranio-spinal nerve-trunk or in an autonomic nerve-trunk, as represented in the diagram.

Ascending and Descending Degeneration.—The nutrition of the neuraxon or nerve-fibre is governed by its originating nerve-cell. When a nerve-fibre is divided (or injured) the part separated from the cell of origin rapidly degenerates (Wallerian degeneration). The nutritional dependence of the nerve-fibres on their cells of origin

FIG. 141.—CRANIAL AND SPINAL PROTONEURONS.   (After Dr. Harry Campbell.)

is one of the leading facts in neuropathology, and it accounts for those *descending* (motor) and *ascending* (sensory) degenerations which we shall meet wit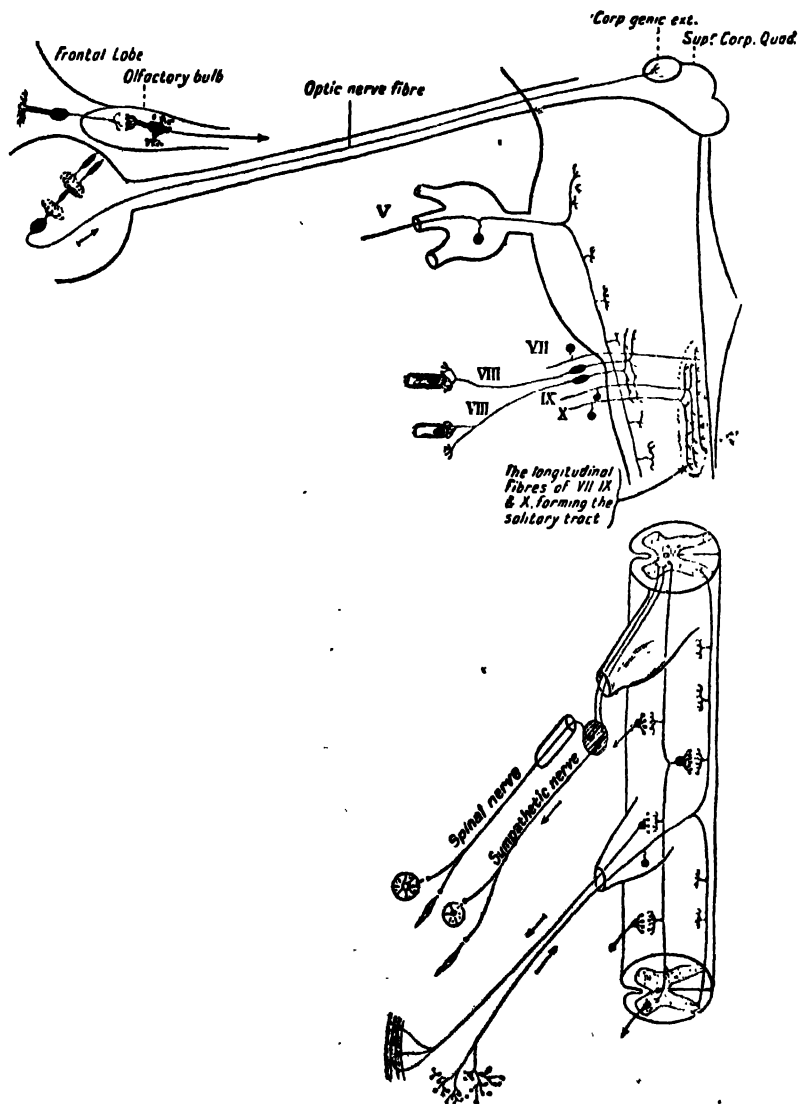h so frequently (Figs. 148 and 149). Thus when a motor cell in the cerebral cortex is severed by injury or disease from the long neuraxon which passes from it down the spinal cord,

a descending degeneration takes place in the pyramidal tracts of the brain and the spinal cord. Conversely whenever the sensory nerve-cells, in a posterior root-ganglion, for instance, are injured, an ascending degeneration takes place in the posterior columns.	•

§ 532. The Brain and Spinal Cord.—Looked at in its simplest form, the cerebrum consists of a mass of white fibres (the corona radiata) spreading out towards the grey cortex. The latter is amplified by means of foldings (convolutions) which in men are more numerous than in any other of the vertebrata (Figs. 143 and 144). Unlike the spinal cord, the grey matter of the brain is found chiefly upon its surface. But in the interior, at its lower part, there are three conspicuous masses of grey matter which, from before backwards, are the corpus striatum, the optic thalamus, and the corpora quadrigemina (two on each side). The corpus striatum is divided into two grey masses (the caudate nucleus internally and the lenticular nucleus externally) by an important band of white matter, the *internal capsule*, which carries the conducting strands from one side of the brain to the opposite side of the body.

As regards *Function* (Figs. 145 and 146) the anterior third, or frontal portion, is largely concerned with intellectual processes. Gross lesions in this position may exist for a long time without any symptoms other than dulness and stupidity, headache, and perhaps vertigo and vomiting.[1] The middle portion just in front of the fissure of Rolando forms the motor area. The different centres for movements of the face and limbs on the opposite side are shown in Figs. 145 and 146. It will be observed (and this will enable us to remember the position of these centres) that the most complex movements, those of the lips and tongue, are farthest *forward* and lowest down, nearest the frontal or intellectual region. Behind and above this in order come the centres for the face, arm, leg, and (on the median aspect of the hemisphere) the trunk. This order, it will be observed, is that of less and less complexity of movement. These centres are not absolutely defined, but overlie each other. The area containing these centres is called the motor area because (1) electrical stimulation of it in animals gives rise to movements, (2) irritative lesions give rise to convulsions, and (3) destructive lesions cause paralysis.

DISEASES OF THE BRAIN may be classed under circulatory lesions, tumours, inflammatory lesions, degenerative lesions, and functional diseases. Vascular lesions (hæmorrhage, embolism, thrombosis) are of sudden, tumours of more gradual, onset. Functional diseases include hysteria, epilepsy, insanity, and neurasthenia. Syphilis may affect the brain in four ways—endarteritis (leading to thrombosis), meningitis, gummata, and neuronic degeneration, as in paralytic dementia. Gummata constitute one of the most frequent forms of cerebral tumours.

Medulla and Spinal Cord.—Those parts of the bulb and the pons varolii which form the floor of the fourth ventricle and the iter a tertio ad quartum ventriculum, contain a series of grey nuclei which give origin to the cranial nerves from the third to twelfth inclusive. These form, as it were, a continuation upwards of the anterior and posterior horns of the grey matter of the spinal cord. Indeed, if we imagine the spinal cord to be split from behind, and as it is traced upwards, to be opened outwards, so that the posterior grey cornua come to be external, and the anterior columns come to the surface beside the middle line, we should find, what is actually the case, that the *motor* nuclei of the twelfth, eleventh, seventh, sixth, and the fifth cranial nerves (motor nuclei corresponding to the anterior horns) lie immediately on each side of the middle line, and that the sensory nuclei of the tenth, ninth, eighth and fifth nerves (corresponding to the posterior horns) lie in a more external situation. The nuclei of

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[1] Apathy and somnolence were absolutely the only symptoms in a case shown by Dr. Ferrier at the Neurological Society in 1892, and in two cases under my own care at the Paddington Infirmary, in 1892, verified by autopsy. These symptoms occur in monkeys deprived of their frontal lobes, but they sometimes recover completely, and regain their faculty of attention.

the other motor nerves (fourth and third), lie farther forward beneath the *iter*. *All the nuclei of the cranial nerves but the fourth pair lie in the floor of the fourth ventricle, while the nuclei of the third and fourth pair lie in the iter* (aqueduct of Sylvius).

Functions.—The bulb contains important reflex and automatic centres. The *reflex centres* are for the closing of the eyelids, sneezing, coughing, sucking, mastication, secretion of saliva, swallowing, vomiting, pupillary movements, and vaso-motor regulation (general dominant reflex centre). The *automatic* centres are respiratory, cardio-inhibitory, vaso-motor, sweating, and convulsion centres. The convulsion centre is situated just where the medulla joins the pons; stimulation of it causes general spasms. This centre may be excited by a venous condition of the blood, as in asphyxia, anæmia (as when the carotids are tied), or congestion, as by compression of the veins coming from the head.

Upper Motor
—Neuron

Intercalary
Neuron
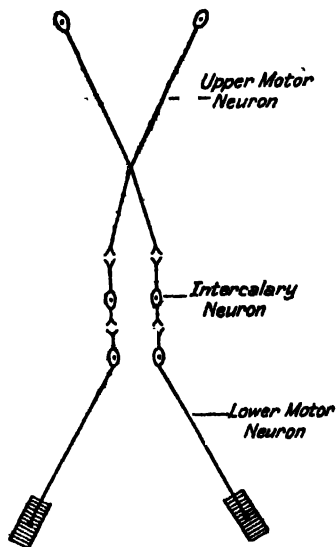
Lower Motor
Neuron

SCHEMA OF MOTOR TRACT.

FIG. 142.—It will be seen by this diagram that the two motor tracts consist each of an upper and a lower motor neuron, associated together by an intercalary neuron. Destructive lesions of the upper neuron cause paralysis, rigidity, and increased deep reflexes. Destructive lesions of the lower motor neuron cause paralysis, atrophy of the paralysed muscles, and altered electrical reactions.

The spinal cord in the adult extends from the margin of the foramen magnum to a point opposite the first lumbar spine, and measures 16 inches. At birth it extends to the bottom of the spinal canal. There are enlargements in the cervical and lumbar regions, corresponding to the nerve supply of the upper and lower limbs respectively. The white matter of the spinal cord contains long descending and ascending tracts and shorter ascending and descending association fibres. These are the axons of neurons, the bodies of which are situated in the central grey matter and in the posterior root ganglia. These short association tracts constitute the *Fasciculus Proprius* (ground bundles). They are concentrated round the grey matter, but also overlap other tracts. In certain regions they form well-defined bundles (Figs. 148 and 149).

The Motor Tract.—It is important to remember that a motor impulse passing from the cerebral cortex to the periphery must pass through at least two neurons, brought into relation with one another by means of an "intercalary" neuron: (1) The *upper motor neuron*, connecting the cerebral cortex with the grey matter (in the floor of the iter, the fourth ventricle, and in the anterior spinal horns) containing the neuron-bodies of the lower motor neurons; (2) the *lower motor neuron*, the axon of which, enveloped in its medullary sheath and neurilemma, constitutes a voluntary motor nerve-fibre. A motor impulse starting in the motor area of the cortex passes through the corona radiata in the central white matter of the brain, through the anterior two-thirds of the hind limb of the internal capsule—through the middle two-fifths of the anterior aspect of the crus cerebri of the same side, through the pons in a band lying between the superficial and deep transverse fibres, and through the anterior pyramids of the medulla. (This description applies to fibres on their way to the spinal nerves.) Here the bulk of the motor fibres cross to the opposite side to form the *crossed pyramidal tract* in the lateral columns of the spinal cord. This tract diminishes in size from above downwards as the axon terminals come into relation with the lower motor neurons in the anterior horns. At the point of decussation in the medulla a few of the motor neuraxons, instead of crossing over, pass down

the same side of the cord in the anterior column close to the anterior fissure, forming the *direct pyramidal tract*, the fibres of which all eventually decussate. There is thus a complete decussation of the descending motor tracts (see Fig. 142). The neuraxons of the lower motor neurons (the spino-muscular level) pass out through the anterior

Fissure of Rolando.
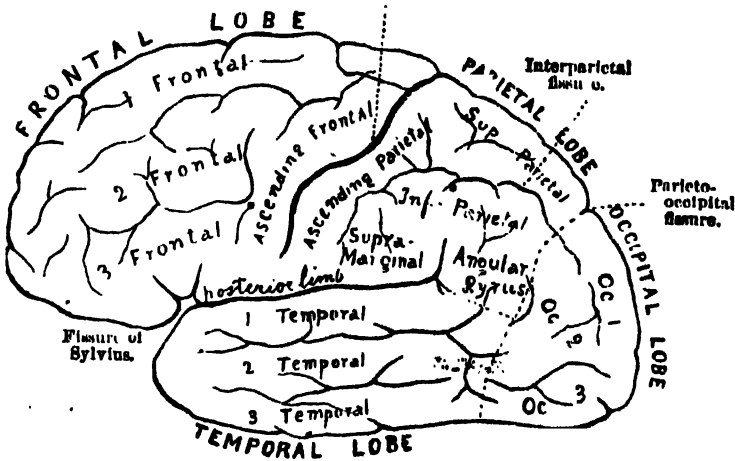
FIG. 143.

Calloso-marginal fissure.

Fissure of Rolando.
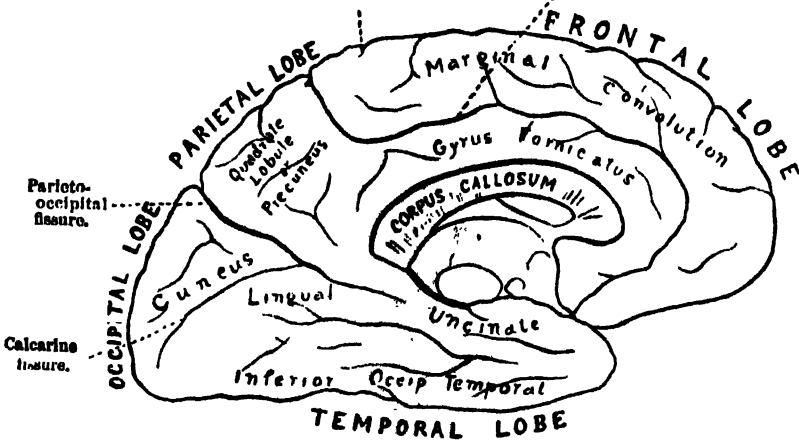
FIG. 144.

FIGS. 143 and 144.—CONVOLUTIONS AND FISSURES OF THE BRAIN, external surface (upper figure) and median aspect (lower figure) of the left cerebral hemisphere. The uncinate lobule is sometimes called the gyrus hippocampus. This and the gyrus fornicatus together form the falciform lobule. The paracentral lobule is the posterior part of the marginal convolution.

nerve roots (or the cranial nerves) into the peripheral nerve trunks, and terminate in the muscles. The total number of fibres (neuraxons) of the lower level passing out through the anterior roots is manifestly far greater than the number of neuraxons in the upper level; it follows therefore that one neuraxon of the cerebro-spinal series

must control several associated neurons of the spino-muscular level. The motor cranial nerves arise from nuclei situated in the iter and the floor of the fourth ventricle. The motor fibres of the cranial nerves all decussate at different levels in the pons and medulla.

Taking a *horizontal section of the internal capsule* (Fig. 159, p. 832), the motor fibres in the genu supply from before backward the eyes, face, and tongue. The motor fibres in the anterior two-thirds of the posterior limb of the capsule supply from before backwards the shoulder, arm, hand, trunk, and leg.

DESCENDING SCLEROSIS.—The cortical motor cells preside over the nutrition of the neuraxons as far down as the grey matter of the lower motor level (in the iter, fourth ventricle, and the anterior horns), and consequently a disease of the cortical cells, or a lesion severing their neuraxons, will be attended by a degeneration (**descending sclerosis**) along the anterior and lateral column to the ends of these neuraxons. The cells in the grey matter of the lower motor level similarly preside over the nutrition of the peripheral motor nerve fibres, and disease of these cells, or a lesion cutting• them off from the neuraxons below, is followed by degeneration of the motor nerves and atrophy of the muscles with which they are connected.

Paralyses due to lesions of the upper motor neurons, situated anywhere between the cortex and the grey matter of the lower level, are attended by muscular rigidity, with increase of deep reflexes, but without muscular wasting, so long as the lower neuron remains intact. Consequently, all hemiplegic lesions and paraplegic lesions attended by lateral sclerosis are recognised by the presence of MUSCULAR RIGIDITY, ABSENCE OF MUSCULAR ATROPHY, and INCREASE OF DEEP REFLEXES in the paralysed area.

The cells of the lower neurons preside over the nutrition not only of the motor nerves, but also of the muscles which they supply, and consequently lower motor neuron lesions are characterised by MUSCULAR FLACCIDITY, ATROPHY, LOSS OF DEEP REFLEXES and ALTERED ELECTRICAL REACTIONS.

Other descending tracts (see Fig. 148) besides the pyramidal (cortico-spinal) are : the rubro-spinal (ventral to the lateral pyramidal) connecting the red nucleus with the ventral horns ; and three tracts running in the anterior columns, namely (from within outwards) : the vestibulo-spinal (from Deiter's nucleus), the tecto-spinal (from the roof of the mid-brain, especially the superior corpus quad.), and the olivo-spinal. The first of these subserves vestibular reflexes, and the second, visual (and auditory ?) reflexes. The function of the olivo-spinal tract is unknown.

§ 533. Sensation and the Sensory Tract.—The neurons constituting the sensory tract are arranged in three levels, a primary or peripheral, a secondary, and a tertiary. The neurons of the primary level extend from the tissues wherein pain is felt to sensory grey nuclei situated in the dorsal spinal horn and (in the case of the 5th nerves) bulb ; and (in the case of neurons subserving the more intellectual sensations) in the nucleus gracilis and nucleus cuneatus. From these primary stations a second series of neurons pass to the optic thalamus. The tertiary sensory neurons are of two orders : the thalamo-thalamic, wholly situated in the optic thalamus, and the thalamo-cortical, which send their axons to the cerebral cortex (see Fig. 147).

Fig. 148 shows the long sensory tracts in the spinal cord. Impulses subserving the more intellectual sensations of discriminating touch (exact localisation, appreciation of compass-points and texture) and active muscular resistance (e.g., estimation of weight by the unsupported hand) pass up the dorsal columns to the nucleus gracilis and nucleus cuneatus in the medulla, and thence by a secondary series of neurons to the opposite optic thalamus, and finally by a tertiary system of neurons to the

cerebral cortex—chiefly to the post-central gyrus (Fig. 147). Impulses subserving the less intellectual sensations of crude touch and pressure, and of temperature and pain, enter the grey matter of the dorsal horns, and are carried thence by a secondary series of neurons to the opposite side of the cord, the former passing up the ventral columns, the latter up the lateral columns, thence to the opposite optic thalamus,

FIG. 145.

FIG. 146.

FIGS. 145 and 146.—LOCALISATION OF THE CHIEF FUNCTIONS on the cerebral cortex of the RIGHT HEMISPHERE, outer surface above, medium aspect below.—The *motor area* was formerly located both in front and behind the fissure of Rolando, but Sherrington's and Grünbaum's researches (Trans. Path. Soc. Lond., 1902), confirmed by Alfred Campbell and others, have shown that it lies wholly in front of the fissure. Moreover, *Common Sensation* is now located behind the fissure of Rolando (from " The Diagnosis of Nervous Diseases," by Purves Stewart).

and finally by a tertiary system of neurons (thalamo-thalamic) to terminate in the optic thalamus (3a, Fig. 147). After interruption of the thalamo-cortical neurons (those *i.e.*, subserving the more intellectual sensations), crude touch, pressure, temperature, and pain sensibility are preserved, but the more discriminating intellectual aspects of sensibility (for which the co-operation of the cerebral cortex is needful) are lost.

It will be seen from this arrangement of the sensory tracts that a lateral hemi-section of the cord will cause (in addition to the motor paralysis of the same side) loss of discriminating touch and muscular sensibility on the same side, and of the crude sense of touch and pressure, as well as pain and temperature sensibility, on the opposite side.

1. The EPICRITIC SYSTEM of fibres run in the cutaneous nerves. They transmit impulses subserving tactile sensation and the discrimination of *slight* differences of temperature. Interruption of these abolishes: (*a*) Recognition of light touch (*e.g.*, by cotton-wool); discrimination of size of object and the compass-points; (*b*) cutaneous localisation; and (*c*) discrimination of temperature between about 25° and 40° C. They subserve in short the higher intellectual aspects of cutaneous sensibility.

2. The PROTOPATHIC SYSTEM of fibres also run in the cutaneous nerves, but by fibres independent of the preceding. They transmit impulses subserving painful cutaneous sensations and *extremes* of temperature. Interruption abolishes: (*a*) Cutaneous pain (*e.g.*, by pricking or strong faradic currents); (*b*) cutaneous temperature sense below about 20° C., and above about 45° C. These fibres are the first to regenerate after section of a cutaneous nerve.

3. The DEEP SENSIBILITY SYSTEM of fibres run chiefly in the nerves to the muscles. They transmit deep **pressure** sensations and **muscular** sensations. These are not destroyed by dividing all the cutaneous sensory nerves. Loss of deep sensibility abolishes: (*a*) The more discriminating tactile, muscular and arthritic sensations; (*b*) sense of pressure (position and degree) in the deep parts. (*c*) Vibration sense. Normally a sense of vibration is felt if a low-pitched tuning-fork is set in vibration and placed on the surface of a bone.

The endogenous (intra-spinal) fibres not only conduct impulses pertaining to these various forms of sensibility, but further establish connections (largely through the medium
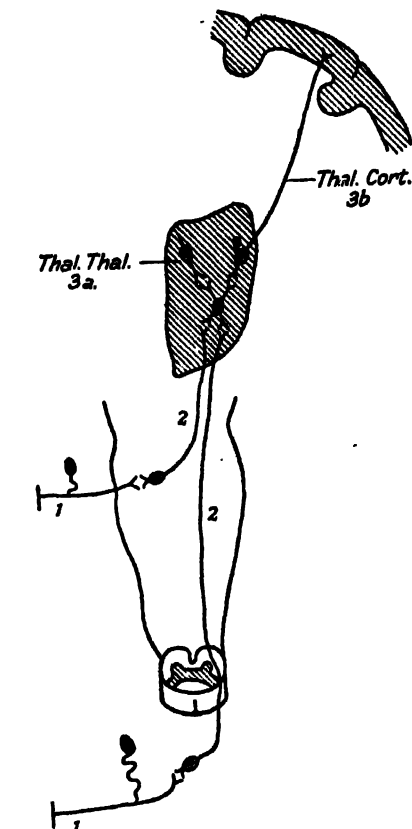

FIG. 147.—Diagram showing the tracts for pain and temperature sensations. The protoneurons after entering the bulb and spinal cord establish immediate connections with the secondary system of neurons whose axons decussate and terminate in the optic thalamus. Here, by means of intercalary neurons, they are placed in relation with tertiary neurons in the thalamus and in the cortex cerebri. This latter arrangement holds good for all the sensory tracts. 1. The protoneuron. 2. Bulbo- and spino-thalamic tract. 3a. Thalmo-thalamic tract. 3b. Thalamo-cortical tract.

of intercalary neurons) for spinal reflexes, as well as with the spino-cerebellar tracts.

§ 534. The Cerebellum.—The cerebellum is situated immediately behind the pons and medulla. It is roofed over by the tentorium cerebelli, so that a tumour or abscess in the cerebellar chamber gives rise to a considerable local augmentation of intra-cranial pressure.

It consists of a central "vermis" and two lateral hemispheres, but modern anatomists speak of an anterior primitive, and a more recently evolved posterior portion, separated by the primary sulcus.

Each hemisphere contains a dentate nucleus and two subsidiary nuclei, while the
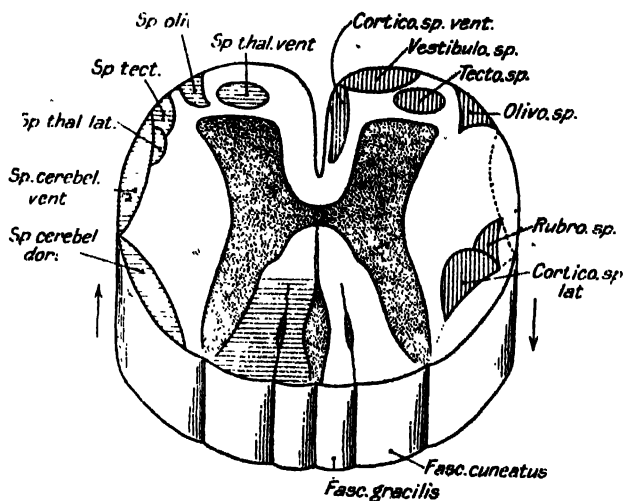
FIG. 148.—(After Dr. Harry Campbell.) Shows on the left the long ascending tracts (horizontally hatched), and on the right the long descending tracts (vertically hatched). The unshaded portion (*Fasc. proprius*) is occupied by short ascending and descending fibres which connect different levels of the cord. These fibres also encroach upon the long tracts. The dark areas in the dorsal fasciculi (columns) are occupied by short ascending and descending fibres of the protoneurons.

FIG. 149.—(After Dr. Harry Campbell.) Shows ascending spinal tracts subserving sensation.

vermis contains the roof nucleus. The cerebellar cortex is receptive : it receives all the cerebello-petal fibres (save those from the vestibular nerves which go to the roof nucleus), while its efferent fibres all terminate in the intrinsic nuclei, which give origin to all the cerebello-fugal fibres.

Besides these *intrinsic nuclei* are certain highly interesting *para-cerebellar nuclei*, the significance of which the student should endeavour to understand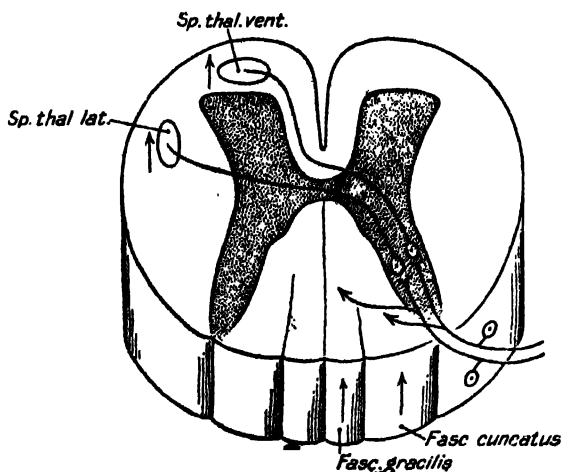. Their interest lies in their close association with the vestibular nerve. They are, on either side : the vestibular nucleus proper, Deiter's nucleus and the nucleus of Bechterew (closely associated with the roof nucleus). The vestibular nerve as it enters the medulla divides into two branches. One branch enters the vestibular nucleus, whence the vestibular tract proceeds, through the optic thalamus, to that part of the cerebral cortex which subserves the sense of the position of the head and the sensation of giddiness. The other branch passes to Deiter's nucleus and the nucleus of Bechterew. These nuclei are placed in relation with the cerebro-spinal motor nerve-nuclei by means of an ascending (posterior longitudinal bundle) and descending (vestibulo-spinal tract) tract. The former is associated with the cranial motor nerve nuclei (including those of the ocular muscles), the latter with the anterior spinal horns.
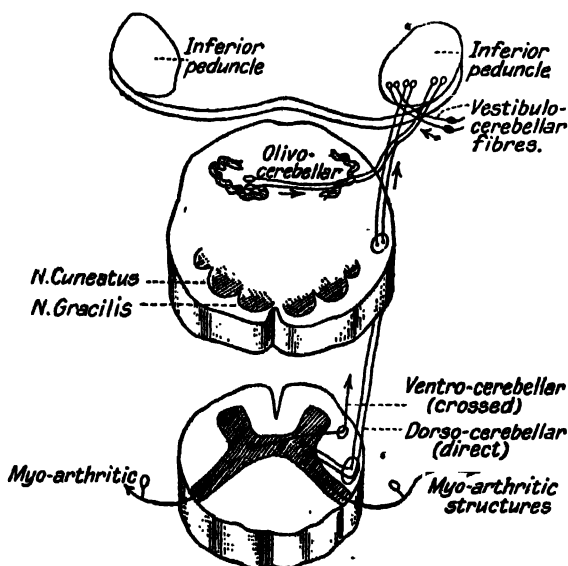

Fig. 150.—(After Dr. Harry Campbell.) Shows the cerebello-petal tracts traversing the inferior peduncle. The ventro-cerebellar fibres enter by the superior peduncle ; the cerebro-cerebellar by the middle peduncle.

Great interest attaches to these nuclei by reason of their myotonic function, which is normally held in check by centres situated higher up in the nervous hierarchy. This myotonic action is shown by the fact that division of the brain-stem at the level of the superior corpora quadrigemina gives rise to decerebrate rigidity, in which the head is retracted and the trunk and limbs maintained in a rigid condition, the elbows being flexed, the wrists pronated, and the lower limbs extended. This rigidity, as Sherrington has shown, persists *even after removal of the cerebellum*, which Hughlings Jackson regarded as its source as well as the source of " tonic fits." It disappears, however, after division of the vestibular nerves, showing that it is reflexly maintained by impulses proceeding from the labyrinth to Deiter's nucleus, and thence down the vestibulo-spinal tract. Stimulation of the anterior surface of the cerebellum has a similar effect.

It is now held that the red nucleus takes a prominent part in the production of decerebrate rigidity, through the medium of the rubro-spinal tract.

The mechanism through which decerebrate rigidity is induced is of capital

importance in the maintenance of the standing position. It also helps to explain the production of certain pathological rigidities. Thus disease, such as tumour, hæmorrhage, meningitis, and internal hydrocephalus, by dissociating the cerebral cortex from the mechanism in question may give rise to total or partial decerebrate rigidity.

The function of the cerebellum is to co-ordinate the motor activities of the voluntary muscles, not merely, as some of the physiology books lead one to suppose, of those pertaining to equilibration, but of all movements requiring co-ordination, such as those of the eyes, articulation, and the manifold delicate movements performed by the hands.

The cerebellar cortex differs from the cerebral cortex in that the layers are the same in every area, the elaborately branched Purkinje cells constituting a prominent feature. From this it is evident that all parts of the cerebellar cortex exercise a kindred function. By observing the effects of stimulating and destroying limited areas of the cortex it has been possible to map out the cortex into definite areas, each of which is related to and controls a definite group of muscles. The dentate nucleus and other intrinsic nuclei through which the cortex controls the muscles have been mapped out in a similar way.

The afferent strands of the cerebellum are mainly three (Fig. 150), and end almost solely in the cortex.

1. The ascending cerebellar tracts, dorsal (through the inferior peduncles) and ventral (through the superior peduncles). These conduct myoarthritic impulses and end entirely in the cortex.

FIG. 151.—Cerebello-fugal tracts. (After Dr. Harry Campbell.) Those passing by the inferior peduncles to Deiter's nucleus are not represented.

2. The vestibular tract from the vestibular apparatus in the inner ear, through the vestibular nerve and the inferior peduncle. This tract conducts impulses which have to do with the position of the head. It differs from the other tracts in that it sends fibres to an intrinsic nucleus—the roof nucleus (as well as the cortex ?). The vestibular nerve, besides sending fibres to the cerebellum, sends fibres to Deiter's nucleus and the nucleus of Bechterew (see Fig. 176), and through these nuclei vestibular myoclonus can be effected independently of the cerebellum. Division of the vestibular nerves causes hypotonus, and removes decerebrate rigidity which, as we have seen, is not removed by excision of the cerebellum.

A considerable band of olivo-cerebellar fibres enter the cerebellum through the inferior peduncle, but the origin and function of this tract is not known.

3. The cerebro-cerebellar tract. This tract passes from the cerebral cortex to the pontine nuclei of the opposite side, entering the cerebellum through the middle peduncle. Of the cerebellar peduncles the middle is by far the thickest. The abund-
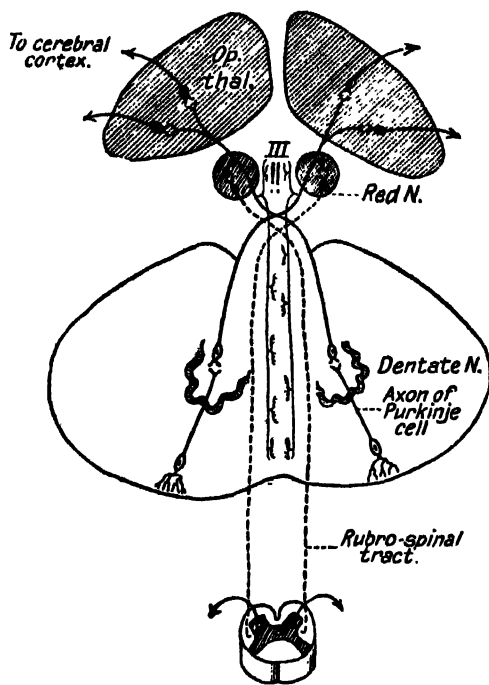
ance of fibres contained in this tract in man is shown by the great diminution in the human brain stem at the lower (caudal) end of the pons.

1 and 2 pass mainly to the vermis, and are probably largely engaged in equilibration. 3 passes mainly to the lateral hemispheres, and is probably mainly concerned with the co-ordination of highly complex voluntary movements such as articulation and the movements of the fingers. It is noteworthy that as the animal scale is ascended the lateral lobes increase in size.

The efferent or cerebello-fugal tracts (Figs. 151) take their origin in the intrinsic nuclei, and it is through these nuclei that the cerebellar cortex influences muscular co-ordination. These tracts leave the cerebellum :—

1. Through the inferior peduncles passing thence to Deiter's nucleus and the nucleus of Bechterew, through both of which the cerebro-spinal motor nuclei are reached.

2. Fibres leave by the superior peduncles and ascending to the level of the inferior colliculi cross to the opposite side. These fibres lead off into three tracts (Fig. 151).

(a) Some pass through the red nucleus to the optic thalamus, whence a further series of neurons send their axons to the cortex cerebri.

(b) Some terminate in the red nucleus whence the rubro-spinal (or pre-pyramidal) tract originates. This tract undergoes rapid decussation (Forel's Decussation) and is placed in relation with the anterior spinal horns. The close association of the red nucleus with the cerebellum helps to explain the frequent occurrence of inco-ordination in connection with lesions of this nucleus.

(c) Other fibres again are placed in relation with the cranial motor nerve nuclei, those associated with the ocular motor nuclei being of special interest. Some of these came off before the decussation. For purposes of simplicity these are not represented in Fig. 151.

§ 535. The Basal Ganglia. Optic thalamus and Corpus striatum.--These centres are philogenitically and embryologically much older than the cerebral cortex. In the lower vertebrates they represent the higher centres of the brain, the optic thalamus constituting the receptive centre for afferent and the corpus striatum the centre for regulating efferent impulses. In the higher vertebrates these centres become subordinated to the cortex cerebri.

In man the optic thalamus contains centres which subserve the crude sensibility of touch, pain, temperature, and vibration, all of which are retained or even increased after severance of the thalamo-cortical fibres. Lesions of the optic thalamus may cause loss of emotional expression by the facial muscles.

Lesions of the corpus striatum may give rise to rigidity or tremor—conditions which are apt to be associated. The rigidity due to disease of the corpus striatum, unlike that which results from disease of the pyramidal tract, does not give rise to increased deep reflexes, ankle clonus or the toe phenomenon. Five diseases are known to occur as the result of disease of the corpus striatum :

(a) Paralysis agitans ; (b) the progressive lenticular degeneration of Kinnier Wilson ; (c) Huntington's chorea, in which the smaller cells of the corpus striatum are affected ; (d) primary or double athetosis—the Vogt syndrome. In this the larger as well as the smaller cells of the caudate nucleus and the putaman are involved ; (e) encephalitis lethargica ; here also the corpus striatum is said to be affected.

§ 536. The Membranes of the Brain and Spinal Cord should be mentioned together, as they are identical in structure, continuous with one another, and subject to the same diseases.

The membranes of the brain and cord are subject to a great many lesions. (1) Meningitis is inflammation of the membranes ; several different forms are recognised. (i.) External or pachymeningitis begins and predominates in the dura. (ii.) Internal or lepto-meningitis begins and predominates in the pia and arachnoid. (iii.) A specific epidemic form of meningitis is recognised, due to the pneumococcus of Fraenkel. (iv.) A septicæmic form is apt to complicate scarlatina and other acute diseases. (2) Tubercle is one of the commonest diseases affecting the meninges in children. It involves the pia mater and arachnoid, and almost invariably starts and predominates

in the transverse fissure and fissure of Sylvius.   (3) Syphilitic meningitis is the commonest meningeal disease in adults.   Syphilis also produces simple thickening of the meninges and disease of its arteries.   (4) Cancer chiefly affects the dura mater secondarily to cancer in other parts of the body.   (5) Fibrous thickening of the dura is found as a chronic form of pachymeningitis.   (6) Bony plates may develop as the result of a chronic meningitis.   I have generally found them in the arachnoid, and chiefly in old people.   (7) Hæmorrhage may take place into both the meninges of the brain and cord ; it may be extradural, subdural, or subarachnoid.   (8) Finally, the meninges of either brain or cord may be the seat of injury.

In all these forms of meningitis the inflammation may start or predominate either in the cranial or spinal meninges, but it is very apt to spread to the other.   It must also be remembered that the superficial parts of the brain and spinal cord derive their nutriment almost entirely from the pia which invests them, and therefore disease of this membrane impairs their nutrition.

●   § 587. The Cerebral Circulation.—The ARTERIES of the brain are derived from the internal carotids and vertebrals, which form the circle of Willis (Fig. in § 588).   The middle cerebral is the most important artery of the brain.   Figs. 152 and 153 show the vascular areas supplied by the cerebral arteries.

VEINS AND SINUSES.— The venules collect the blood into veins of the brain (which have no valves).   These enter the cerebral sinuses in a direction opposite to the blood-flow.   Most of the blood leaves the cranial cavity through the internal jugular veins, which are continuations of the lateral sinuses.   Should they be blocked, a few collateral communications, through which the intracranial circulation communicates with the veins outside the skull, are available.   Thus (1) the anterior end of the superior longitudinal sinus communicates with the veins of the nose ; (2) the ophthalmic veins communicate through the orbit with the facial veins ; (3) the lateral sinus communicates with the occipital vein through the mastoid cells ; (4) the superior longitudinal sinus communicates through the calvarium with the veins of the scalp ; and (5) the inferior petrosal sinus communicates with the deep cervical veins.

The veins of Galen collect the blood from the collateral plexus of the lateral ventricles, and empty themselves into the straight sinus, and thence into the torcular Herophili and through the lateral sinus into the internal jugular vein.   Pressure on, or thrombosis of, these veins tends to produce distension of the lateral ventricles with fluid (hydrocephalus).

The PECULIARITIES OF THE CEREBRAL CIRCULATION are as follows : The brain is supplied by *cortical* arteries from the pia mater and the *central* arteries which, coming off from the circle of Willis and its neighbourhood, penetrate the great ganglia at the base.   (1) There are no anastomoses between the cortical and the central arteries, nor between the individual central arteries among themselves ; and only very few between the several cortical branches.   (2) There are no communications between the several branches of the vertebral, basilar, and cerebellar arteries, which supply the pons and bulb ; but the superior, middle, and inferior cerebellar arteries communicate freely.   Hence the greater frequency of softening in the former as compared with the latter positions.   (3) The cranium being a rigid box, the brain incompressible, and the quantity of intra-cranial cerebro-spinal fluid always being much the same, the quantity of intra-cranial blood is always much the same, but its velocity varies considerably, and the velocity depends largely on the blood-pressure in the carotids and vertebrals.   This pressure, among other factors, is regulated by the amount of blood in the splanchnic area.   When this is dilated, as happens in sleep, the general blood-pressure is low, and the circulation in the brain is slow ; conversely, when the splanchnic area is contracted, the general blood-pressure is raised, and the circulation in the brain becomes more rapid.   Dr. Leonard Hill maintains that the vaso-motor regulation of the cerebral arteries is feeble.

§ 588. The Autonomic (Involuntary, Vegetative) Nervous System.—This system controls the activities of the involuntary muscle-fibres and gland-cells.   It consists

C.M.

of two portions having different functions. These we may speak of as the *sympathetic* and the *para-sympathetic*. (See Figs. 154, 155.)

Autonomic fibres leave the central nervous system as medullated pre-ganglionic fibres ("connector" fibres of Gaskell). These terminate in ganglia, where they are placed in relation with secondary autonomic neurons. These give origin to non-medullated post-ganglionic fibres, which are distributed to muscle- or gland-cells. (It is possible that in some cases, at least, a tertiary neuron is intercepted between the post-ganglionic fibre and the tissue-cells.)
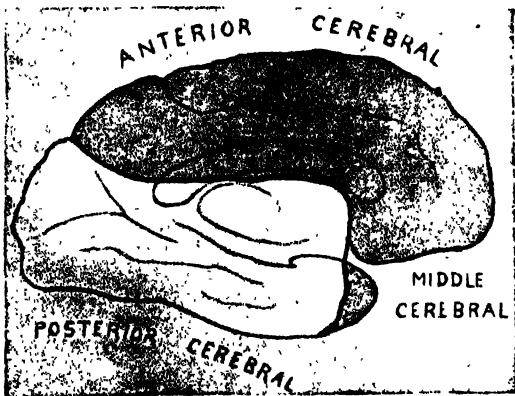
FIG. 152.
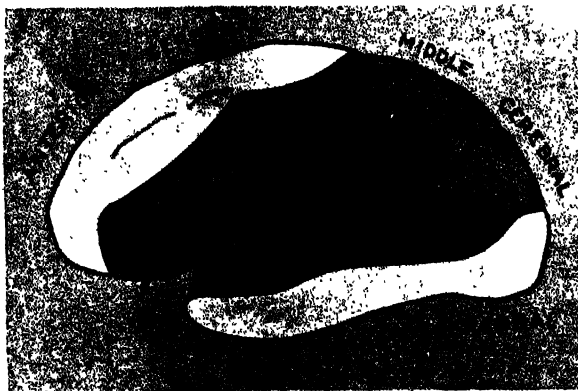
FIG. 153.

FIGS. 152 and 153 show the areas of distribution of the CEREBRAL ARTERIES—median surface of the brain above, external aspect of the brain below.

The outflow of the sympathetic fibres. These leave the central nervous system by the twelve pairs of thoracic nerves, and the 1st and 2nd pairs of lumbar nerves.

The outflow of the para-sympathetic fibres. These issue by the 3rd, 7th, 9th, 10th and 11th cranial pairs (the autonomic fibre of the 11th pass, however, into the 10th), and by the 2nd and 3rd sacral pairs; the latter give origin to the *pelvic nerves.*

The sympathetic supplies practically all the involuntary muscles and most of the gland-cells in the body. The para-sympathetic supply, on the other hand, is limited to the viscera (e.g., the iris, the lachrymal glands, the parotid, and the thoracic and

Op.
tthal.

Lent.
N.

3. To ciliary gang.
7. To Meckel's and sub-max. gang.
9. To otic gang.
10. To the cardiac and other visceral ganglia.

Cervical Sympathetic

Sympathetic Ganglionic Cord

1. Thoracic
2.
3.
4.
5.
6.
7.
8.
9.
10.
11.
12.

1. Lumbar
2

2 Sacral
3

Parasympathetic fibres from the 2nd and 3rd sacral to form the pelvic nerve
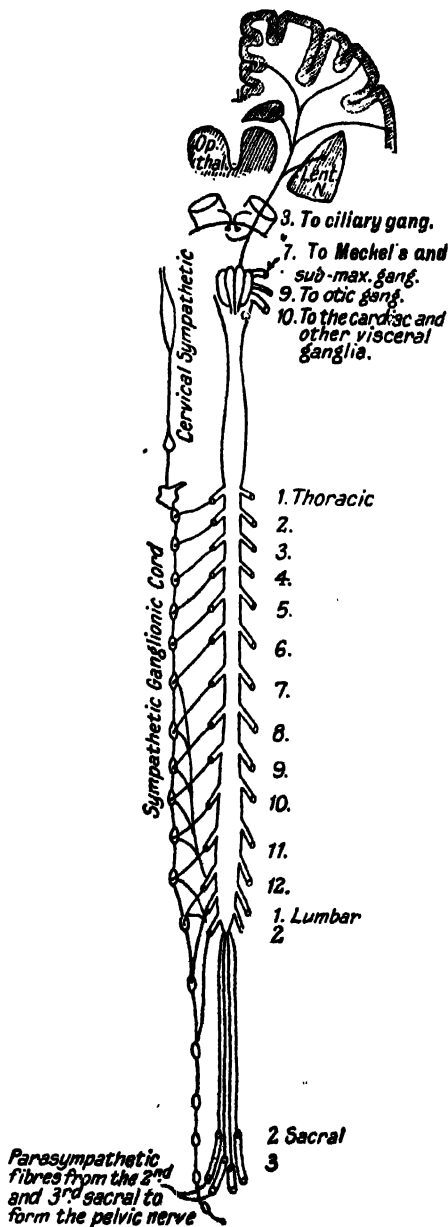
FIG. 154.—(After Dr. Harry Campbell.)

This diagram shows the exit of the autonomic nerves from the central nervous system. The sympathetic fibres issue by the twelve thoracic and first two lumbar nerves. The para-sympathetic fibres issue by the third, seventh, ninth and tenth cranial nerves, and by the second and third sacral nerves. The representatives of the autonomic system in the cerebral cortex and 1   ganglia are also indicated.

Ant. root
Mixed nerve
White ramus
Ganglion
Grey ramus
Pre-ganglionic fibre to viscus.
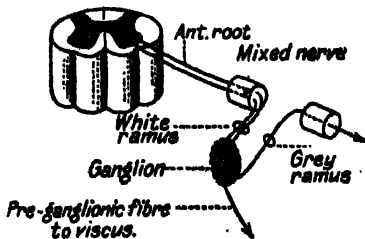
FIG. 155.—(After Dr. Harry Campbell.)

This diagram shows pre-ganglionic white fibres issuing from the spinal cord by an anterior spinal root, and passing thence into the mixed nerve trunk, white ramus, and ganglion of the sympathetic chain ; one of these pre-ganglionic fibres is seen passing on to a viscus, the other is placed in synaptic connection with a post-ganglionic neuron. This gives origin to a post-ganglionic grey fibre which enters the mixed nerve viâ a grey ramus.

abdomino-pelvic viscera), which thus have both a sympathetic and para-sympa-
thetic supply. These two supplies exercise a reciprocal action. Thus the sympathetic
augments, the para-sympathetic (vagus) inhibits, the heart-beat. The sympathetic
retards the propulsive action of the stomach, intestines, and bladder, causing relaxa-
tion of the propulsive fibres and constriction of the sphincters (pyloric, ilio-colic, anal
and vesical), while the para-sympathetic (vagus and pelvic) has the opposite effect,
stimulating the propulsive fibres and relaxing the sphincters. The sympathetic
supply to the viscera is effected mainly through the splanchnics, while the sympa-
thetic fibres to the head, neck, trunk, and limbs run in the cranial and spinal nerves
as post-ganglionic fibres. The pre-ganglionic fibres of the sympathetic issue from
the mixed nerve-trunks (all the thoracic and the 1st and 2nd lumbar) as the white
rami communicantes. Those going to the mixed nerves terminate in the sympathetic
chain-ganglia. From the latter post-ganglionic fibres issue as grey rami (post-gang-
lionic fibres), which, entering the nerve-trunks, are distributed to the tissues; pre-
ganglionic fibres passing to the abdominal viscera terminate in ganglia beyond those
of the sympathetic chain, where they are placed in connection with post-ganglionic
neurons.

The chromaffin cells in the medulla of the adrenals represent the primitive sympa-
thetic system as met with in the leech, and it is remarkable that the adrenalin which
they secrete has the same effect upon muscle- and gland-cells as stimulation of the
sympathetic nerves. When such stimulation is required adrenalin is poured out
into the venous spaces surrounding the chromaffin cells, thus gaining access through
the blood to the sympathetic nerves.

The pre-ganglionic fibres of the para-sympathetic system are distributed with the
nerves (cranial or sacral), by which they leave the central nervous system to ganglia
situated peripherally. Thus pre-ganglionic fibres are conducted by the vagus to the
cardiac ganglia, from which post-ganglionic fibres pass to the cardiac muscle-fibres.

It is probable that the peripheral sensory neurons of the autonomic system are
akin to ordinary protoneurons.

§ 539. General Principles in Neuro-pathology.—(1) When the nutrition of a neuron
is impaired, *the peripheral endings of* its processes are the first to show degeneration.
An illustration of this is seen in tabes, in which the peripheral termination of the
sensory muscle neuraxon (muscle spindle) may be the first to undergo degeneration.

(2) *Prolonged forced functioning of any nerve structure results in its atrophy.* This
is sometimes known as Edinger's principle, but it is one with which neurologists have
long been familiar. A typical instance of it is seen in the tremor, spasm, and atrophic
paralysis which accompany various occupation neuroses. It helps to explain why the
light reflex is so frequently lost in tabes, and why the extensors of the wrist suffer
most in the lead palsy of painters.

(3) *Ascending and descending scleroses are a consequence, not a cause, of the atrophy
of the neurons* (the essential tissues). If one tissue element is removed—by degene-
ration or otherwise—the surrounding tissues (deprived of this opposition) tend to
take on increased activity, growth, and proliferation.

(4) *Functions which are last acquired in the evolution of the nervous system are the
first to succumb to disease, and vice versâ.* An illustration of this principle is seen in
mental disorders, in which the comparatively recently acquired moral sense is early
disordered and in the loss of memory for recent events observed in old people. Again,
in aphasia (loss of memory for the symbols of thought) the memory for printed or
written signs is lost more often and sooner than the memory for spoken words.

PART A. SYMPTOMATOLOGY

The symptoms of diseases of the nervous system may be *subjective* or
objective. Among the objective symptoms defects in muscular power
are the most obvious. These are considered seriatim under Paralysis,

Inco-ordination, and Disordered Gait, Muscular Rigidity, Tremor, and Amyotrophy, in Parts B. and C. **Defects of sensation** and the **special senses** are dealt with there also.

The *subjective symptoms* met with in disorders of the nervous system are very numerous, but they can practically all be brought under one of six headings—**defects of the mental powers, nervousness, disordered sleep, vertigo, pain,** and other **disordered sensations.**

The **mental powers** are often disturbed. Such disturbances include inaptitude for mental work, transient disturbances of thought, stupor, or coma, loss of speech or of memory, restlessness, excitement, or delirium, or more chronic perversion of the mind, amounting to insanity (§§ 572 to 577).

It is useful to remember that delirium, convulsions, and headache are evidences of *cerebral irritation* ; whereas mental dulness, stupor, paralysis, and coma suggest rather *cerebral compression* or intense toxæmia.

§ 540. **Nervousness** is a symptom which frequently guides us to disorders of the nervous system. In its colloquial sense it generally means " easily agitated." The patient comes to us, for instance, because the least noise startles him or the least worry upsets him. (1) It is a leading symptom in *neurasthenia* (§ 556) and disorders of the endocrine system.[1] (2) *Hysteria* is another common form of nervousness ; here we have an inherent emotional and vaso-motor instability, which is manifested by "hysterics" and various other forms of attack.[2] (3) In many *structural diseases* of the nervous system, particularly those mentioned under the symptom tremor, the patient comes to us for what he calls nervousness. (4) Chronic alcoholism, morphinism are manifested by nervousness ; also (5) convalescence from severe illness, and other conditions of debility.

§ 541. **Pain and Neuralgia.** —Pain is a common symptom of disease, but not more common in diseases of the nervous system than of other systems, unless the peripheral nerves are involved. A careful observer may derive considerable help by investigating the four important features of pain : (1) Its position ; (2) its character—whether throbbing, pricking, shooting, knife-like, dull, aching, etc. ; (3) its degree ; and (4) its constancy—*i.e.*, whether persistent or intermittent, or a combination of the two (exacerbating). We should never allow ourselves, for the sake of time or trouble, or by pandering to the ignorance or whim of our patients, to treat pain simply as pain, by the administration of soporifics, hypnotics, and the like. Pain is a sure indication of abnormal function, and we should endeavour to trace out its *cause*, however difficult the task. The best method of investigation is, first, to examine the suspected nerve and its immediate neighbourhood for direct causes of irritation, and particularly any bony orifice through which it may pass ; secondly, to seek for any *reflex cause* of irritation in organs more or less distant, such as the teeth, and the uterus ; and, thirdly, to search for any general or *constitutional*

[1] " Clin. Lects. on Neurasthenia," fourth edition, Glaisher and Co., London, 1909.
[2] " Lectures on Hysteria," Glaisher, London, 1909.

derangement which may act as a predisposing or exciting cause, such as anæmia, rheumatism, gout, tubercle, or syphilis.

PAIN and NEURALGIA in different parts of the body and limbs are discussed under Neuralgia (§ 641).

§ 542. **Headache** (Cephalalgia) is the commonest of all pains. The position of the pain is not, in my experience, much guide to its cause.

The *Causes* of headache are numerous. Inquiries should be directed to possible LOCAL, GENERAL, and REFLEX causes, 'as in the case of pain and neuralgia.

(*a*) Among the following LOCAL CONDITIONS, the first five cause continuous pain of some duration. (1) *Syphilitic disease of the cranium* is a frequent cause of continuous headache ; a marked feature of this headache is its nocturnal exacerbation and associated tenderness. (2) Various *meningeal conditions*, acute or chronic, cause pain, and here syphilis again may play a leading part. When children under three show signs of headache the possibility of tuberculous meningitis should be kept in mind, and the temperature promptly taken. (3) *Intracranial tumours*, especially when affecting the meninges or cortex, may be known by the association of vertigo, occasional vomiting (especially when the headache is worst), and optic neuritis ; localised tenderness over the seat of the lesion is sometimes observed. (4) *Ear disease* causes headache, and in such cases, pressure over the mastoid cells often reveals tenderness. (5) Disease of the *frontal sinuses*, usually secondary to nasal or post-nasal catarrh, gives rise to dull, continuous headache. (6) The *supra-orbital branch* of the fifth nerve supplies the forehead, and neuralgia of this nerve produces frontal headache of a shooting and paroxysmal character. (7) *Excessive brain work* is frequently followed by a feeling of dull, heavy weight on the vertex. (8) The wearing of hard and heavy hats, and (9) *Pediculi capitis* are other causes of headache.

(*b*) Among the CONSTITUTIONAL or GENERAL CONDITIONS the first seven causes mentioned below are due to toxic or hæmic conditions, and perhaps the eighth and ninth also. Headaches of this class are apt to undergo periodic exacerbations. (1) *Chronic interstitial nephritis* should always be suspected in the elderly, a suspicion which is confirmed when the patient frequently rises at night and passes large quantities of urine of low specific gravity. (2) *High blood-pressure* is probably in operation in the foregoing, but from whatever cause arising it is frequently associated with headache. (3) *Hepatic derangement* and the condition known as *lithæmia* give rise to what is sometimes called bilious headache. *Constipation*, and derangement of the stomach are amongst the most frequent causes of headache in everyday life. (4) *Chronic alcoholism, gout, rheumatism, syphilis*, and *plumbism* may also be attended by headache. The syphilitic headache is so severe as to interfere with work, and is clinically of great importance in that it may herald an outbreak of cerebral syphilis, *e.g.*, hemiplegia, and thus calls for vigorous anti-syphilitic treatment. (5) *Malaria*, rarely seen now in this country, is attended by a severe

frontal headache : hence the term " brow ague."   (6) *Pyrexia* is usually accompanied by headache. (7) *Chlorotic patients* suffer a good deal from headache. (8) Closely allied to the preceding is the headache of *exhaustion* or inanition. (9) Headache is frequent in hot, unventilated rooms. (10) *Hysteria* and *neurasthenia* are often attended by headache. The first is sometimes likened to a nail being driven into the skull at one spot (" clavus ") ; the second frequently takes the form of a feeling of constriction around the head or pressure on the vertex (" casque neurasthenique " of Charcot).   (11) *Migraine*, which is a special paroxysmal form of headache, is described in § 642.

(c) REFLEX CAUSES.—(1) *Eye-strain* is an extremely common cause of headache in modern times, and is generally associated with some uncorrected error of refraction. It may arise without any such error in those who read small print too much. (2) Diseases of the nose, teeth, and auditory apparatus (especially the two first) are potent causes of reflex headache. (3) In *diseases of the viscera*, especially of the heart, lungs, uterus, and liver, a pain referred to the head is often present.

The palliative *Treatment* of headache as a symptom will be found under Neuralgia and Migraine, §§ 641 and 642.

§ 543. Disordered Sleep.—In regard to sleep we have to rely very much upon a patient's own account, and some care is required in accepting his statement in this matter. Persons differ considerably in the amount of sleep they require. The aged will do with half the sleep of adolescents, and the middle-aged with half that of babyhood, the respective quantities being approximately five and ten hours, eight and sixteen hours. The popular belief that " six hours for a man, seven for a woman, and eight for a fool " is sufficient sleep is altogether wrong. Sir William Jones' adaptation in the eighteenth century of an old Persian saying- –

> " Seven hours to work, to soothing slumber seven,
> Ten to the world allot, and all to Heaven,"

perhaps comes much nearer the truth.   .

Sleep may be (a) DEFICIENT in quantity (insomnia), (b) DEFECTIVE in quality (restlessness, dreaming, etc.), or (c) EXCESSIVE in quantity.

(a) INSOMNIA, wakefulness, or deficient sleep may arise under the following conditions : (1) All *painful affections* are apt to be attended by sleeplessness. (2) *Defective hygienic conditions* or mode of life—*e.g.*, late suppers in those unaccustomed to them, indigestible food, mental overwork, especially just before bedtime, worry, a strange or uncomfortable bed, cold feet. (3) In neurasthenia, hysteria, and other functional *disorders of the nervous system*, mental excitement, all acute and many chronic forms of mental derangement, sleeplessness may be one symptom of the ailment. (4) In the absence of any of the foregoing causes, search should be made for some *general constitutional condition*. Sleeplessness, like head-ache, in the elderly should always make one suspect chronic interstitial nephritis. Such patients often complain of " cat-sleeps "—*i.e.*, dropping

off for a few minutes at a time. (5) Among *local disorders*, cardiac disease, causing the patient to start up as soon as he falls off to sleep with a feeling of suffocation, may be mentioned.

(*b*) DEFECTIVE SLEEP. —(1) *Dream-disturbed sleep* generally indicates a toxæmia of some kind. *Nightmares* and *dreams* in the young are often due to an undigested meal taken late. They are also a characteristic symptom of neurasthenia, and various other toxic conditions of the blood. (2) *Night terrors* in children are sometimes induced by dietetic or gastro-intestinal defects—especially in nervous children. Among other causes may be mentioned nasal or pharyngeal obstruction (*e.g.*, adenoids). Pro-tracted night-terrors should make us suspect petit mal, especially when combined with nocturnal incontinence. There is a residue of cases in which no cause is apparent excepting the neurotic diathesis, which sub-sequently becomes manifest by the development of hysteria, epilepsy, chorea, neurasthenia or by mental degeneracy.[1] (3) *Sleep-walking* and sleep-talking are curious phenomena in which certain functions of the brain are not only active, but sometimes in an exalted condition. (4) *Twitching of the limbs* as a person drops off to sleep is a common symptom of some toxæmias (particularly those of intestinal origin), or hypersensitive-ness of the nervous system (such as occurs after morphinism, § 560).

(*c*) EXCESSIVE SLEEP.—Drowsiness is apt to accompany the intense venous congestion of *heart disease* ; it is also an early symptom of *urœmia* and occurs in other *toxic states*. I was once consulted by a curate who went to sleep almost as soon as he sat down in church during certain parts of the service, or even when his rector was preaching. In that case it was apparently due to *disordered liver*, which being remedied, the sleepiness disappeared. Undue sleepiness after a meal always indicates disordered digestion. In *hysteria*, attacks of sleep (narcolepsy) may supervene at unexpected times. Drowsiness is a frequent symptom of brain tumour. The *trypanosoma* produces " sleeping sickness," in which the patient sleeps for days, and then generally dies. It is a characteristic feature of Encepha-litis Lethargica. Persistent and even fatal sleep has been known to follow *influenza*. Cases have been recorded in which sleep has lasted for several days at a time, and in which the attacks were prevented by the adminis-tration of *thyroid*.[2]

The *Treatment* of insomnia must be directed to the cause. The first essential is to raise the general health to the highest possible level, and to inculcate tranquillity of mind : a person who is in sound health and who does not worry sleeps soundly. Extrinsic causes of insomnia, such as noise, a hard bed, too high or too low a pillow, too little or too much bed-clothing, an ill-ventilated bedroom, should be removed ; also intrinsic causes, such as pain, pruritus, frequent micturition, cough, nasal obstruction. Hypnotism is sometimes of value. Among the simpler remedies, a cup of warm milk or gruel, or a hot bath or hot-pack, last

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[1] See a clinical lecture by Dr. Leonard Guthrie, *Clinical Journal*, June 7, 1899.
[2] Dr. Lewis Bruce, *Scottish Medical and Surgical Journal*, December, 1910.

thing at night, is often useful. Dyspepsia after the evening meal must be carefully treated. Sometimes an evening walk is useful, or Swedish and deep-breathing exercises, or similar means to take the mind away from the occupations of the day. Massage or cold compresses over the legs or abdomen may promote sleep. Sod. hypophosphite, 20 grains (1·3), in warm milk at bedtime is a simple yet excellent remedy for the sleeplessness of mental fatigue; or bromide of ammonium, 20 grains (1·3), thrice daily, or an alkaline draught of 50 grains of bicarbonate of soda in a tumbler of hot water. A weak galvanic current through the brain or high-frequency currents are sometimes found beneficial. When these simpler measures are unavailing, recourse (with due safeguards) may be had to hypnotics and sedatives such as the following : Alcohol, chloral hydrate, camphor, cannabis indica, hyoscyamine, lupulin, paraldehyde, bromidia, chloralamide, chlorobrim, sulphonal, veronal, trional, chloretone, bromural and last but not least dial-ciba.

§ 544. **Vertigo** is a sensation as if the body were rotating, or as if objects wore rotating round the body. It is accompanied by a momentary loss of equilibrium which may cause the patient to reel or actually fall down. It is sometimes described as a "giddiness," a "dizziness," or "swimming in the head." The rotation may appear to be horizontal, vertical, or oblique ; or the patient may feel that he is rising into the air or sinking into the ground. The sensation is often attended by nausea and vomiting as indeed intense giddiness may be from whatever cause arising.

The *Causes* of vertigo are : (a) Diseases of the ear, (b) diseases of the nervous system, (c) diseases of the eye, (d) diseases of the circulatory organs, (e) affections of the stomach (gastric vertigo), and (f) laryngeal spasm.

(a) AURAL VERTIGO.—The first thing to do in any case of vertigo is to decide whether it be accompanied by defect of hearing. Any disease of the external meatus, Eustachian tube, or middle ear, which is accompanied by alteration in the pressure of the fenestra ovale, is apt to be attended by giddiness and defective hearing. Impacted wax may suffice to cause it. It may result from syringing the ear. The deafness, which may be slight, may be either *obstructive deafness*, such as arises in chronic middle-ear catarrh, or *nerve deafness*, such as that from labyrinthine disease (see Menière's disease, § 563).

(b) DISEASES OF THE NERVOUS SYSTEM.—(1) In *epilepsy*, vertigo often constitutes the aura or warning of the convulsive attack (grand mal) and the whole phenomenon in attacks of minor epilepsy (petit mal). With this form of vertigo there is usually a temporary interruption of consciousness. (2) *Hysteria. neurasthenia*, or the depressed condition of the nervous system produced by drugs, alcohol, or mental strain, may be accompanied by giddiness. Hysterical vertigo generally results from the patient being startled or frightened. (3) *Intracranial lesions* such as tumour, especially when affecting the cortex and the vestibular nerve, may give rise to giddiness. Vertigo is a prominent symptom in lesions of the *cerebellum* or its peduncles, and here giddiness and a reeling gait are often the leading and sometimes the only symptoms. (4) *Disseminated sclerosis* is attended in three-fourths of the cases (Charcot) by vertigo. (5) A disease has been described under the name *endemic paralytic vertigo*, occurring in Switzerland and Japan, characterised by paroxysmal vertigo and paralysis. (6) A sudden vascular lesion of Deiter's nucleus produces acute vertigo, tinnitus, deafness, nausea, nystagmus, and sometimes pain in the distribution of the fifth nerve.

(c) In regard to CIRCULATORY DISORDERS (1) giddiness constitutes the first stage of syncope. Poverty of blood, anæmia, convalescence, or exhausted states of the system, may give rise to giddiness. Anæmia is perhaps the commonest cause of it in the young. (2) Any *cardiac weakness* may give rise to vertigo, especially among

the aged. Excessive smoking, by acting as a cardiac depressant, may act in the same way. (3) *Arterial disease* is attended by vertigo, which even more than the preceding is characterised by coming on chiefly when the patient rises from a sitting or stooping posture. This Senile Vertigo, or as I have elsewhere described it,[1] *postural vertigo*, very often occurs on rising at night to pass water, or on getting up in the morning, or any sudden alteration of posture (and see § 562). (4) Vertigo in the aged may be the only indication, at the time of their occurrence, of the *minute hæmorrhages* or *softenings* so often found in their brains after death. (5) *High blood-pressure*, temporary or permanent, is not infrequently attended by giddiness. Vertigo in the aged should always be taken as a warning of some kind of cardiac or vascular failure, and the circulatory system should be carefully investigated in such cases.

    (d) OCULAR VERTIGO is usually due to an error of refraction, or fixation (especially when so marked as to cause diplopia). In this case it is relieved by shutting one or both eyes. (So also is the dizziness felt on climbing a height, or in a wide open space, as in the agoraphobia of psychasthenia.) Among refractive errors marked astigmatism is perhaps the commonest cause of vertigo. It is also apt to occur in myopia owing to the constant strain of the internal recti.

    (e) GASTRIC VERTIGO has been described by several observers (*e.g.*, by Trousseau), but I believe many such cases are of circulatory origin. The close relation between the taking of food and the giddiness, to which some refer, is sufficiently explained by the effect of a full stomach upon the heart. It is certain that the toxæmia produced by gastro-hepatic disorders may cause giddiness, especially on rotating the eyes strongly in one direction.

    (f) TOXIC VERTIGO. Toxæmia is a potent cause of vertigo, such as alcohol and nicotine poisoning, and poisoning from disordered states of stomach, liver, and bowel.

    (g) A LARYNGEAL VERTIGO has been described in which the patient on attempting to cough gets giddy and may fall.

    The *Treatment* of vertigo must be directed to the cause, but as a palliative measure the bromides are of great value.

Disordered Subjective Sensations of many kinds may be complained of in diseases of the nervous system, and are mentioned in their proper place.

    a. Those referable to the head, such as a feeling of weight or lightness.

    b. Those referable to the *extremities*, such as numbness and tingling.

    c. Those referable to the *special senses*, smell, sight, hearing, and taste.

    d. Those referable to the *mind*, hallucinations, delusions, etc.

    e. Those which affect the *equilibrium* are mostly included under the term vertigo or giddiness (*vide supra*).

PART B. CLINICAL INVESTIGATION

§ 545. There are *two problems* in the diagnosis of nervous cases : first, to LOCALISE the lesion (Is it generalised, or in the brain, the cord, or the nerves ?) ; and, secondly, to ascertain its NATURE. The following is a scheme for a systematic and complete investigation :—

First, examine the leading sign or symptom.

Secondly, inquire into the history of the present illness, the past personal history, and the family history.

The **History** of the case is of considerable importance, and must be patiently and thoroughly investigated.

As regards the *history of the present illness*, many nervous symptoms are vague, or what the patient regards as unimportant. Sometimes the onset is acute, but more

    [1] *British Medical Journal*, January 23, 1897, and Transactions of the Pathological Society of London, 1904.

frequently it is insidious and chronic. Many nervous diseases run a prolonged and changing course, and the physician who sees a case three or four years after its onset may have to depend for his diagnosis wholly or mainly upon the history of the case. The exact dates (1) of ceasing work, and (2) taking to bed are important.

The *previous history* may reveal gout, tuberculosis, syphilis, lead, or other metallic poisoning. Alcohol and syphilis play a wide rôle in the etiology of nervous diseases. The toxins of diphtheria and influenza among the infective fevers are highly inimical to the nervous system. Traumatism, mental and emotional strain often play a part in the etiology. The influence of sexual abuses is considerable, though apt to be exaggerated. Some diseases, like hysteria and migraine, are recurrent, and the history of previous attacks is an important aid to diagnosis.

In the *family history* a neuropathic diathesis may be revealed. In a nervous family the inherited instability may take the following forms : epilepsy, migraine, hysteria, neurasthenia, eccentricity, psychasthenia and insanity. Consanguineous marriages intensify this diathesis.

Thirdly, proceed to the examination of—

    I. The general symptoms and mental attitude, temperature, pain.

    II. The muscular system—motor power, co-ordination, gait, spasm, tremor, atrophy.

    III. The deep and superficial reflexes.

    IV. The electrical reactions.

    V. The special senses and cranial nerves.

    VI. The cutaneous sensibility.

    VII. The organic reflexes ; the trophoneuroses ; the autonomic system and the angioneuroses.

    VIII. The cerebro-spinal fluid in certain cases.

§ **546. General Symptoms.**—The majority of cases met with in private practice and out-patient work belong to the **generalised neuroses,** in which the symptoms consist of vague pains or bodily discomfort, nervousness, mental depression, restlessness, insomnia, etc. In many such cases an *examination of all the organs and functions of the body* will reveal some defect to which the nervous condition is secondary. The neurologist needs to be a skilled general physician.

The patient's MENTAL STATE plays an important part in all disorders of the nervous system. We have to judge how much of the malady exists in the patient's mind, how far we can rely on his description of his symptoms. Some of the chief points to investigate are sleep, dreams, memory, reasoning power, decision, attention, moral standard, delusions, hallucinations. In judging a person's mental condition one needs to be a man of the world first and a physician afterwards. This was what Sydenham meant when he replied to a father who asked what books his son should read as a preparation for the medical profession—" Let him read ' Don · Quixote.' " Common sense and tact as well as experience are indispensable. It is, moreover, necessary to enter sympathetically into the feelings and thoughts and mode of life of your patient before he will give you his entire confidence.

Pyrexia is absent in most chronic diseases of the nervous system. It ushers in Infantile Paralysis, and attends inflammatory affection such as

Meningitis. In cerebral abscess it may be absent, excepting just at the outset. Persistent fever in a case of nervous disease suggests some inflammation of the cerebro-spinal meninges.

Pain and its method of investigation are dealt with under Neuralgia (§ 641), and in various other parts of this work.

The investigation of the *skull* is considered in §§ 13 and 664.

§ 547. The **Muscular System** affords some of the most reliable evidences of disease of the nervous system. The muscles may be affected in four ways. Thus there may be—

    (a) WEAKNESS or paralysis.

    (b) INCO-ORDINATION.

    (c) Muscular SPASM (tonic or clonic), or TREMOR.

    (d) ATROPHY.

Muscular defects may also reveal themselves by alterations in the *reflexes* (superficial and deep) and in the *electrical reactions*.

(a) Is there any **loss of power**? Can the patient walk? Can he sit up in bed? Can he move each of his limbs as a whole? Is the motor weakness localised to a few muscles? The *degree* and exact *position* of the weakness should then be investigated. Paralysis is a total, paresis a partial, loss of power. In the case of the handgrip the degree of weakness can be measured by a dynamometer, but in other instances we can only roughly estimate it by the degree of resistance on the part of the operator necessary to prevent a particular movement.

HEMIPLEGIA is paralysis of one side of the body; PARAPLEGIA, paralysis of both legs; MONOPLEGIA, paralysis of one limb; DIPLEGIA, paralysis of both sides of the body; BRACHIAL DIPLEGIA, paralysis of both arms; DIPLEGIA FACIALIS, of both sides of the face. In states of coma it is often difficult to test the presence of paralysis; its existence on one side may be indicated by a greater limpness on that side—the arm when raised and allowed to drop falling inertly—or by the fact that it can only with difficulty be raised on account of the rigidity on the paralysed side. Sometimes an *individual muscle* or *a group of muscles* is affected, and a knowledge of the action of muscles enables us to decide which is involved. Go patiently through the movements of individual joints, *offering passive resistance* to each movement in turn; in this way we can determine the extent and the degree of the paralysis. The big joints are capable of six movements—flexion, extension, adduction, abduction, rotation, and circumduction; the smaller joints only the first two, or the first four. The action and nerve supply of the various muscles are given under plexus and single nerve paralysis (§ 605).

(b) Is there any **inco-ordination** or **alteration of gait**?

INCO-ORDINATION is a defective co-operation of the different muscles involved in a purposive movement, unaccompanied it may be by any loss of muscular power. In the *legs* inco-ordination is manifested by an exaggeration of the normal movements (as in locomotor ataxy), or an inability to balance while walking (as in cerebellar tumour). The patient may be asked to walk along the edge of the carpet. If in bed ask him to follow your finger round in a circle with his big toe, or to touch, with his eyes closed, the dorsum of one foot with the big toe of the other. Another test is to get him to stand with toes and heels together and the eyes shut, observing if he stands steadily or sways about (ROMBERG'S SIGN). A very delicate test of the same kind is to ask him to balance himself on tiptoe, with knees bent and eyes closed. Note should

always be made whether a patient can or cannot walk, and he should, if possible, be made to walk before us. The *gait* in locomotor ataxy, paralysis agitans, spastic paraplegia, and many other affections, is characteristic (§ 613). To test the *upper extremities* ask the patient to thread a needle, or (with eyes shut) to bring his two forefingers tip to tip in front of him, or to touch the tip of his nose, button or unbutton his coat. Co-ordinated muscular movement depends upon the integrity of the arthritic and muscular sense (whereby the position of the limb in space is estimated), the vision, and the cerebellar control. When the eyes are closed or bandaged, vision is eliminated.

A common form of inco-ordination or *ataxia* is that due to interruption of the sensory tract, whether of the sensory nerves (alcoholic neuritis), the posterior nerve roots (*e.g.*, radiculitis), or the posterior column (tabes dorsalis). Cerebellar inco-ordination may also occur from interruption of the spino-cerebellar tracts (Friedreich's ataxia), and from a variety of lesions in the neighbourhood of the cerebellum, interrupting the tracts to and from that organ, or from a lesion of the cerebellum itself.

Vestibular Ataxia is induced by stimulating one labyrinth by injecting cold water into one external auditory meatus, or by stimulating both labyrinths either by rotating the patient in a revolving chair, or by passing a galvanic current between the two mastoid processes. If one ear, say the right, be stimulated with cold water, a horizontal nystagmus occurs when the subject looks to the left. If now with closed eyes he attempts to touch with his finger the examiner's finger held in front of him (having previously noted its position with the eyes open) the subject's finger passes by several inches to the right of the target. This mispointing is known as Barany's sign.

The term *muscular sense* is falling out of use. It is better to speak of (1) the sense of passive position, (2) the sense of passive movement, (3) the kinæsthetic sense, or sense of active resistance.

(1) *Sense of passive position.*—This is the sense of the relation to one another of the skeletal segments—*e.g.*, of a limb—which have been put in a given position by the observer, the patient remaining passive, *i.e.*, keeping his muscles wholly relaxed. This sense depends upon impulses from the joints mainly (joint sense), and to a less extent from the muscles and tendons.

(2) *Sense of passive movement.*—This is the sense of movement, and of the direction of movement, of a passively moved part, *e.g.*, a limb. It depends upon the same kind of impulses as the last.

(3) *The kinæsthetic sense.*—This is the sense by which we estimate the strength of muscular contraction needful to overcome a resistance, *e.g.*, to raise a limb, lift a weight, push an object out of position. This sense is not—as was held by Bain—centrally initiated, but (as in the case of the sense of passive movement and position) due to impulses generated in the muscles, tendons, and joints, notably in the muscles. The delicacy of this sense is shown by the fact that it is largely by the kinæsthesia of the ocular muscles that we judge of distance. It is tested by placing objects of the same size and configuration, but of different weights, in the patient's unsupported hand. A match-box containing coins, and another cotton-wool, may be used, or objects can be placed in a handkerchief and slung on to the hand or foot. Normally a healthy person can detect a difference of one-seventeenth between two moderate weights.

(1), (2), and (3) all tend to be lost in tabes. In this disease there may be complete anæsthenia of the muscles, tendons, and joints, the patient when his eyes are closed having no knowledge whatever of the position of the affected part.

(c) Is there any **spasm (tonic or clonic) or tremor** ? If so, note its kind, degree, and distribution. Tonic spasm or rigidity is a continuous muscular contraction ; clonic spasm consists of intermittent muscular contractions. Smaller, more rapid vibratory movements are known as *tremors*.

A *tonic rigidity* is obvious to the observer on attempting to bend the limb ; it may be generalised, as in the case of tetanus, or confined to the paralysed limbs as the

result of descending sclerosis in hemiplegia and paraplegia. Early and late rigidity are referred to under hemiplegia.

Clonic spasms and *tremors* often become more obvious when the patient holds up the affected member. Some tremors are only present when the affected muscles are in action (intention tremor)—*e.g.*, those of disseminated sclerosis.

Athetosis is a peculiar position of slow mobile spasm intermediate between tonic and clonic spasm.

Convulsions are violent clonic spasms. It is important to ascertain—first, whether the convulsions start at one spot, or generally ; secondly, whether they are attended by unconsciousness ; thirdly, whether the fit is preceded by a warning ; fourthly, the patient's condition after the fit ; fifthly, whether there is involuntary evacuation of motions or urine, or biting of the tongue ; sixthly, whether the patient has had any previous attacks of the same or a different kind.

Hypertonia is an increase in the tone of all the muscles hardly amounting to tonic spasm ; *hypotonia* is a diminution of the muscular tone. *Kernig's sign*, which is found in 85 per cent. of cases of acute cerebro-spinal meningitis, is an evidence of hypertonicity, but is not pathognomonic of this disease. There is inability to flex passively the extended lower limb at right angles to the abdomen ; the leg flexes on the thigh before the latter forms a right angle with the abdomen. In these cases it is also impossible fully to extend the leg on the thigh in the sitting posture.

(*d*) Is there any **muscular atrophy** ? Atrophy may be elicited roughly by pinching the muscles and finding them flabby and wasted, or by the measuring-tape. **Lesser defects** of muscular nutrition can only be obtained by electrical examination (§ 549).

Muscular atrophy arises from (1) *disuse* ; (2) diseases of the *motor nerve nuclei* at the base of the brain or in the ventral horns ; (3) diseases of the *ventral roots* of the *peripheral nerves* ; (4) disease of the *motor nerves* ; (5) diseases of the *muscles* (myopathies) ; and (6) diseases of the joints.

§ 548. III. There are three kinds of **Reflexes** to be investigated—**deep, superficial**, and **organic.**

(*a*) Are the **deep reflexes** altered ? When the tendon of a muscle that has been put on the stretch is struck, the muscle immediately contracts and produces a jerk of the limb. This is known as the deep or " tendon reflex." To elicit the KNEE-JERK or PATELLAR TENDON REFLEX get the patient, if possible, to sit on the edge of a bed, table, or chair, with the legs *hanging freely*, or cross one leg over the other and let it hang as though it did not belong to him. These positions slightly stretch the quadriceps extensor and reflexly increase its tone. Now strike the patellar tendon sharply with the tip of the fingers, or the edge of a rubber-shod hammer or stethoscope, and the leg will immediately jerk forwards from the sudden contraction of the quadriceps. The patient's attention may be engaged by conversation or by his hooking the fingers of the two hands tightly together, and trying to pull the hands apart : this is called the " reinforcement " of the knee-jerk. An increase or diminution in the knee-jerk may be conveniently indicated by k.j. + or — 1, 2, or 3. The strength of the knee-jerk varies in health ; it is least marked in the young and in the old. It is exaggerated in all UPPER MOTOR NEURON lesions ; it may also be increased in hysterical and other functional neuro-muscular irrita-

bility.   It is diminished or lost in LOWER MOTOR NEURON lesions and, indeed, when any part of the lower reflex arc, motor or sensory, is interrupted.

The *knee-jerk is increased* (k.j. +) (1) when the pyramidal tracts are affected by sclerosis ; all destructive *upper neuron* lesions lead to such sclerosis.   (2) When there is increased irritability of some part of the reflex arc, as in (i.) tetanus and strychnine poisoning. or (ii.) spinal meningitis.   (3) When there is defective inhibitory control from the higher centres, as in hysteria (strychninism of Charcot), or in toxæmic states, such as phthisis, typhoid, or neurasthenia.

The *knee-jerk is diminished* (k.j. —) *or absent* (1) when the lower reflex arcs belonging to the quadriceps are interrupted either in their afferent or efferent portion (*e.g.*, from lesions of the nerves, nerve roots, or the portion of anterior horn pertaining to these arcs).   (2) When myopathy affects the quadriceps muscle (*e.g.*, pseudo-hypertrophic paralysis).   (3) During coma, and immediately after the convulsive stage of epilepsy.

Most of the *superficial tendons* can be tested in the same way, though not with the same facility, the essential points being to (1) get the patient to relax the muscle to be tested, (2) gently stretch the muscle, and (3) strike its tendon.   The tendo Achillis or *ankle-jerk* may be tested while the patient is kneeling on a chair with the calf relaxed.   The triceps or *elbow-jerk* is elicited by hanging the patient's elbow over your wrist, and striking the triceps tendon ;   the *supinator-jerk* by tapping the tendon just above the styloid process ;   and the *wrist-jerk* by striking the extensor tendons when the hand is hanging loosely.   The *jaw-jerk* is not present in health ;   when present it can be elicited by placing one of your fingers firmly on the front of the chin—the mouth being open—and tapping it as in percussion.

The *osseal*, or *periosteal*, *jerks* are similar to the tendon jerks.   Thus the supinator jerk, just referred to, may be elicited by striking the radius just above the wrist, while the forearm is slightly flexed in a position midway between pronation and supination.

ANKLE-CLONUS, or the clonus imparted to the calf muscles by stretching the tendo Achillis, is elicited by supporting the patient's knee with one hand and suddenly dorsiflexing the foot with the other hand, gently maintaining pressure on the ball of the foot.   It is present in *organic diseases* under the same circumstances as increased knee-jerk and ankle-jerk—*i.e.*, in *upper neuron lesions*.   Under the same conditions *knee-clonus* may be obtained by suddenly pressing the patella towards the foot while the leg is extended, and steadily maintaining the pressure.

The tendon reflexes are of use not only to detect which of those two important groups of lesions is present—upper or lower motor-neuron lesions—but also to ascertain approximately the level of the cord which is involved, as may be seen from the table on next page.

Myotatic Irritability consists of the too-ready contraction of a muscle when its muscular substance is struck.   It is present under the conditions in which the tendon jerks are increased.   It is frequently seen in advanced phthisis, and other exhaustive diseases—*e.g.*, in percussing the chest, but in such cases the contraction is limited to the fibres actually struck.   In tetany the condition is very marked, the facial muscles being thrown into contraction when lightly tapped.

(*b*) **The Superficial Reflexes.**—On stimulation of certain parts of the skin or mucous membrane with a blunt pin or the tip of a penholder, a contraction of certain associated muscles takes place (see table in § 594).   It is a true reflex action, and can only be obtained when the afferent and efferent paths and the corresponding grey matter in the cord or brain are intact.   The chief clinical use of the superficial reflexes is to

TABLE SHOWING THE SPINAL SEGMENT INVOLVED IN THE DEEP REFLEXES.

Reflex.	*Spinal Segment Involved.*	*Opposite Vertebral Spines of—*
Jaw-jerk.	Motor nucleus of the fifth cranial nerve.	
Supinator-jerk.	Fifth cervical.	Third C. spine.
Wrist-jerk.	Sixth cervical.	Fourth C. spine.
Elbow-jerk.	Seventh cervical.	Fourth C. spine.
K.-J.	Second and third L.	Tenth and eleventh D. spines.
Ankle-clonus.	Third, fourth, and fifth sacral.	First S.

determine the locality of a disease in the spinal cord. The mode of eliciting these reflexes and the situation of the spinal centres for the plantar, gluteal, cremasteric, epigastric, abdominal, and interscapular reflexes are given in the table just referred to. The conjunctiva reflex, obtained by touching the conjunctiva, causes contraction of the orbicularis palpebrarum, and its centres are situated in the fifth nucleus (sensory) and the seventh nucleus (motor). The palate reflex, obtained by touching the soft palate, leads to its elevation by the levator palati; the afferent nerve is the ninth, the efferent the accessory part of the element (through the vagus). The cutaneous reflexes vary considerably in different persons. They are more prompt in children than adults, and in women than men, and are difficult to obtain when the skin is harsh and insensitive or when there is much subcutaneous fat. The abdominal reflexes are generally absent in old people.

BABINSKI'S REFLEX is a modification of the plantar superficial reflex. To elicit it the patient should be in the recumbent posture, with the lower limb slightly flexed, and the foot warm and dry ; the sole is gently stroked upwards by the finger-nail or a blunt pin. Normally the big toe and the other toes become flexed upon the sole. In Babinski's reflex there is *extension of the great toe* followed by flexion of the other toes. The stroking should not be hard enough to evoke dorsiflexion of the foot, as this obscures the big toe reflex. After infancy this reaction is only met with in *organic disease* involving the pyramidal tracts. It is best elicited by stroking the outer side of the sole. In *functional* cases the plantar reflex, if it can be elicited, gives a *flexor* response as in health ; in hysteria, local anaesthesia, and a diminution of the plantar reflexes with exaggerated knee-jerks is a characteristic combination. In infancy, before the age of walking, an extensor response is normal.

§ 549. **Electrical Examination of Muscles** and the nerves supplying them is an important aid in distinguishing *lower* from *upper* motor neuron lesions, and in estimating the degree of disease or degeneration in nerve or muscle.

The APPARATUS REQUIRED is not complex. The chief requisite is a *faradic coil*, because the first and most important point is to ascertain the degree of faradic contractility. A simple coil, in which the secondary coil slides on to the primary, is needed. The wire of the former should not be too thin, so as not to cause unnecessary pain. A couple of moderate sized Leclanché cells will drive it. Two wires and three electrodes are required, one large (measuring about 6 by 4 inches), a round medium-sized one, and one small (about 1 inch in diameter). The two smaller ones should be fitted with an interrupting handle.

A suitable *galvanic battery* is more expensive, for it requires at least twenty-four small Leclanché cells (capable of giving about 35 to 40 volts), a collecting board and reverser, with electrodes as before, and, to be complete, a galvanometer that has been properly tested (Fig. 156). The constant current can be used from the main with a suitable switchboard. Recently testing by *condenser discharges* has been found to be most useful.

To *test the faradic response* place the large electrode on the patient's chest, on the back of the neck, or some other indifferent position, and another electrode, connected with an interrupting handle, over the motor point of the nerve or muscle to be tested. If the current is too strong for estimating the finer degrees of difference, the operator should take this second electrode in one or other of his hands, and apply his well-wetted finger to the well wetted skin of the patient. A knowledge of the motor points of nerve and muscles is not indispensable, for they can be discovered by applying the electrode at different points, and noting at what points contraction is most easily obtained. The motor point of a muscle is near the point of entry of its nerve; that of a nerve is generally near its most superficial part. The electrodes and the skin should be very *thoroughly* wetted with plain warm water. Some prefer salt and water,
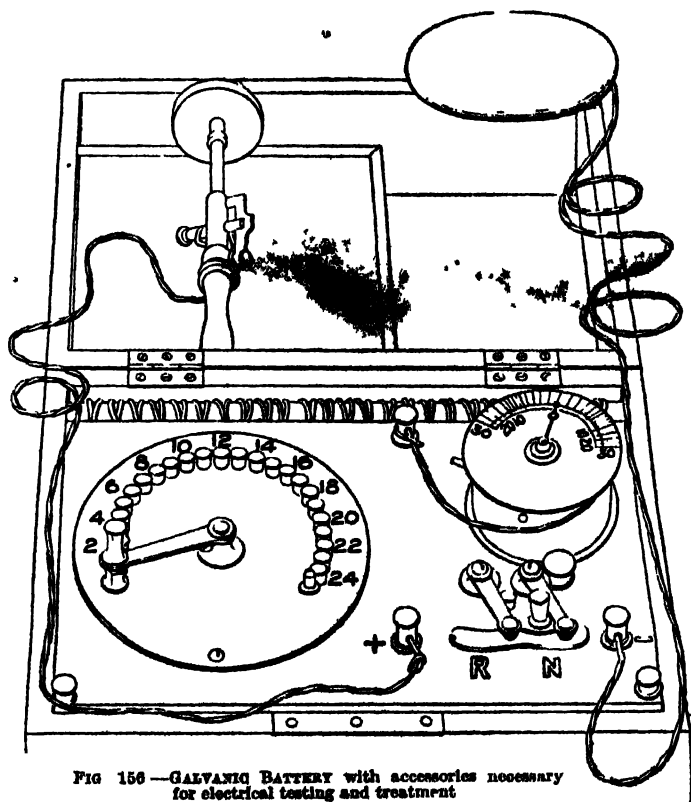
Fig 156 —GALVANIC BATTERY with accessories necessary
for electrical testing and treatment

although this increases the conductivity, it is not necessary, provided the electrodes are kept clean with the use of a little soap. The patient should be placed in a good light so that both sides of the body can be seen equally. Having thoroughly moistened the skin over the part to be tested, as well as the corresponding region of the other side, ascertain first (by gradually sliding up the secondary coil) the *minimum* current necessary to produce a minimum contraction of the muscle or muscles on the healthy side. Then test the side suspected of disease with the same amount of current, to see if the same degree of contraction is produced, or if not what strength of current is requisite.

The faradic contraction of a muscle can only be obtained through its nerve, so

C.M.

that when the nerve is completely degenerated, the muscle (though still contractile to the direct stimulus of galvanism) fails to respond to faradism. Hence when a muscle does not respond to faradism, but does respond to galvanism, we conclude that the nerve is profoundly degenerated, but that the muscle-substance still retains some contractility.

To *test the galvanic reaction* the electrodes are placed in the same position as before. For (a) *quantitative* alterations, compare the two sides as before, noting what amount of current (as indicated by the number of cells used, or the number of ma. registered by the galvanometer) is required to produce a minimal contraction on both sides. (b) For *qualitative* alterations begin with the kathode (negative pole) placed on or about the motor point under investigation. Close the current by means of the inter-rupting handle ; the contraction obtained is known as the Kathodal Closing Con-traction (K.C.C.). (If you cannot tell which is the kathode, place the two wires in a glass of water, and a lively production of hydrogen gas comes off at the kathode ; or place both wires on a piece of wetted litmus paper, which becomes reddened around the positive pole (anode) from the liberation of oxygen. Next convert the electrode on the patient into the anode or positive pole by means of your reverser, and repeat the process of closing the current. The resulting contraction is called the Anodal Closing Contraction (A.C.C.). Normally, with the same strength of current and the same degree of wetting of the skin, K.C.C. is greater than A.C.C. (K.C.C. > A.C.C.), or what amounts to the same thing, a greater strength of current is required to produce A.C.C. than K.C.C.

Muscular contraction with a galvanic current is only produced at the closing or opening of the current. The normal order of the contractions is as follows :

K.C.C. > A.C.C. > A.O.C. > K.O.C. (O.C. = Opening Contraction).

Abnormally A.C.C. is equal to or greater than K.C.C.

The REACTION OF DEGENERATION (R.D.) differs in different stages. When a nerve is severed or is the seat of acute inflammation, after a preliminary increased electrical reaction to both currents during the first two days, (a) the action is gradually lost to both currents *during the ensuing ten days*, the faradic reaction not being regained unless regeneration takes place.

(b) In the *second or third week*, and for some weeks afterwards, the galvanic reaction is restored, and Erb's **reaction of degeneration** occurs in its typical form. It is charac-terised by—

(i.) No muscular contraction to **faradism**, however strong the current.

(ii.) A quantitative increased contraction to the galvanic current.

(iii.) The galvanic contraction, which in health is prompt and sharp, becomes sluggish, and often the response is better when the electrode is placed over the peripheral end of the muscle rather than over the motor point.

(iv.) Qualitative galvanic changes are present in that A.C.C. is equal to or greater than K.C.C.

(c) *Two or three months later* the galvanic contractility gradually disappears (unless regeneration is established), though it may happen that for one or two years A.C.C. can be obtained with a progressively increasing strength of current.

The **diagnostic** value of electricity is considerable, for the R.D. is present in some form or other in all severe lesions of the **lower motor neuron**—the anterior horns, anterior roots or peripheral nerves (motor or mixed)—while it is absent in lesions of the **upper motor neuron**.

List of diseases in which the Reaction of Degeneration is found :

I. Diseases of the *anterior horns* :

    1. Acute and subacute anterior poliomyelitis.

    2. Chronic anterior poliomyelitis (*e.g.*, spinal form of progressive muscular atrophy).

    3. Amyotrophic lateral sclerosis.

    4. Syringomyelia and spinal gliosis.

5. Cervical myelitis, transverse lumbo-sacral myelitis, in the arms or legs respectively.

6. Diseases of the motor nuclei in the medulla corresponding to the anterior horns (progressive bulbar paralysis, acute inferior polio-encephalitis).

NOTE.—In chronic anterior poliomyelitis (progressive muscular atrophy) there is a slow progressive involvement of the anterior cornual "cells" and, therefore, irregular electrical results are obtained, because one part of a muscle may be more involved than another. In idiopathic myopathy irregular results are also obtained.

II. Diseases of the *anterior roots* :

    1. Compression from tumours and thickened membranes (*e.g.*, in syphilis, and hypertrophic cervical pachymeningitis).

    2. Compression in the intervertebral foramina in vertebral diseases (caries, tumours, fracture, luxation).

III. Severe diseases of the *peripheral nerves* :

    1. Trauma or pressure from tumours, persistent thickenings, bone disease, operations, etc.

    2. Neuritis of toxic origin—*e.g.*, lead, alcohol, arsenic, diphtheria, and other infections.

The diagnosis of Myasthenia Gravis is facilitated by finding a characteristic faradic reaction of exhaustion. The muscles at first contract normally to the faradic current, but after a few contractions they get "tired," and will not contract even with the strongest current. This is known as the myasthenic reaction. There is no similar alteration to galvanism.

In regard to prognosis, when the faradic response is obtainable the prognosis is good. Its loss is consistent with recovery, so long as the muscles react to galvanism. Complete loss of galvanic reaction is of bad omen. Slight voluntary movement sometimes returns *before* the return of the electrical reactions.

The slighter the lesion the less the alteration in electrical excitability. Slight compression of a nerve enough to produce paralysis may cause but little alteration, though faradic excitability is generally somewhat diminished.

§ 550. The investigation of the **Special Senses** and **Cranial Nerves** is given in detail hereafter (§ 645). The points to investigate are as follows :

    1st nerve.—Smell.

    2nd, 3rd, 4th, and 6th nerves.—Ocular symptoms : the condition of the vision and fundi, the condition of the extrinsic and intrinsic muscles.

    5th nerve.—Taste, facial sensation, muscles of mastication.

    7th nerve.—Facial muscles, taste.

    8th nerve.—Hearing, tinnitus, vertigo.

    9th, 10th, and 11th nerves.—Pharyngeal sensation, deglutition, muscles of palate and larynx, sterno-mastoid and trapezius, heart, respiration.

    12th nerve.—Muscular power of tongue.

§ 551. **Cutaneous Sensibility.**—Four kinds are now recognised—touch, pain, thermal, and pressure sensibility. We inquire whether these are increased or diminished, and seek to determine the exact area of altered sensibility. To test a person's capacity to feel we must first obtain his intelligent co-operation. His eyes should be covered, and he should be instructed to say simply " yes " immediately he perceives any sensations. Babinski's plan in testing hysterical patients is to ask, " What am I doing now ? " so as to eliminate the element of suggestion. He warns the student against using the phrase, " Do you feel this ? " Corresponding points on the two sides of the body should be tested if possible, and a negative test should be applied from time to time. The sensibility differs in different persons and in different parts of the body. It is

often necessary to make several examinations and to strike an average. The student should first study § 533, the remarks on Epicritic, Protopathic, and Deep Sensibility. In mapping out areas of anæsthesia, begin with the anæsthetic area and work outwards. The reverse plan should be adopted in mapping out areas of hyperæsthesia.

(1) TOUCH sensibility may be tested by the finger or a wisp of cotton-wool. For the investigation of epicritic sensibility the blunt points of compasses are sometimes used. Ascertain whether (i.) sensation is *lost* (anæsthesia) or *increased* (hyperæsthesia), and (ii.) the *boundaries* of such loss or increase. In hysteria, local areas of an an- or hyper-æsthesia may be present, and pressure on the tender spots (hysterogenic zones) may produce fits. (iii.) Tactile sensation may be *delayed*, as in peripheral neuritis and tabes, or (iv.) *misplaced* (allocheiria) as in tabes. *Atopognosis* is failure to locate a sensation properly. *Astereognosis*, or the inability to recognise the shape of an object (*e.g.*, a key) by the feel, is due to defect in the muscle- and joint-sense, as well as of cutaneous sensibility. It has been thought to be due to a lesion in the post-central gyrus. Loss of sensation in the hairs is known as *tricho-anæsthesia.*

(2) Cutaneous sensibility to PAIN may be tested by the sharp end of a pin or by the faradic current. It may be lost (*e.g.*, in syringomyelia) while sensibility to light touch is retained.

(3) Cutaneous sensibility to PRESSURE is tested by means of a blunt object such as a pencil or an algometer.

(4) Cutaneous sensibility to TEMPERATURE (thermal sense) is tested by test tubes of hot and cold water, or hot and cold silver spoons, or by alternately breathing and blowing on the skin. Loss of the thermal sense is a feature of syringomyelia.

(5) Other forms of cutaneous sensibility are ITCHING sensibility (the paths subserv-ing which travel with those of pain and temperature) and the sense of the DIRECTION OF TRACTION of the skin when a fold of it is pulled in different directions.

(6) DEEP SENSIBILITY to PRESSURE is frequently lost in tabes. Muscles may become completely anæsthetic in this disease. On the other hand they may be exquisitely tender in peripheral neuritis. Arthritic sensibility to torsion and the sensibility of the mucous membranes are lost in some cases of hysteria.

(7) The VIBRATION SENSE is tested by means of a low-pitched tuning-fork, placed while vibrating over a superficial bone. This may be lost before other forms of sensibility in tabes. This sense also pertains, though in less degree, in the nails and the connective tissues.

The KINÆSTHETIC and the JOINT SENSE are described in §§ 533 and 547*b*.

Paræsthesia (perverted sensation) and various subjective sensations are described in the disorders of sensation. The term *aura* is applied to the morbid sensation or "warning" which precedes epileptic fits.

§ 552. **Organic Reflexes.**—Defæcation, micturition, deglutition, and respiration are sometimes spoken of as the organic reflexes, because they consist of the contraction of involuntary muscular tissue, resulting from the automatic stimulation of a mucous membrane. As regards *defæcation* and *micturition*, the patient should be questioned as to the existence of retention or incontinence, particularly in cases of paraplegia. Such symptoms indicate direct or indirect involvement of the lumbar enlargement, and frequently occur in myelitis and certain system lesions, such as tabes dorsalis.

§ 553. **Trophic Changes.**—The nervous system controls the nutrition, not only of muscle- and certain gland-cells directly, but indirectly of all the organs and tissues of the body. There is no evidence of the existence of specific trophic nerves. The trophic change which results may be revealed in a variety of ways.

The *skin* may become " glossy "—*i.e.*, smooth, thin, and atrophied. Erythema, ulceration (as in the perforating ulcer of tabes) or sloughing (*e.g.*, bedsores) may appear, vesicles may develop when an irritative lesion of a root-ganglion (*e.g.*, of the fifth cranial nerve) is present, and painless whitlows on the fingers, as in syringomyelia.

The *bones* may undergo absorption or their development be arrested (as in polio-myelitis).

The *joints* may be the seat of painless effusion and bone absorption (as in tabetic arthropathy).

. § 554. Disorders of the Autonomic System have hitherto been but little studied. They may give rise to angio-neurotic œdema, and various disorders of the circulation and of nutrition.   With the exception of those of the veins, the autonomic system controls all the involuntary muscle-fibres in the body (intestinal, pulmonary, cardio-vascular, genito-urinary, cutaneous) as well as most of the gland-cells ; and effects of its disease (unrecognised perhaps) may occur in any organ or tissue.

PART C. DISEASES OF THE NERVOUS SYSTEM : THEIR DIAGNOSIS, PROGNOSIS, AND TREATMENT

- § 555. **Routine Procedure and Classification.**—*First*, as on previous occasions, we ascertain and investigate the patient's LEADING SYMPTOM, probably one of those mentioned in Parts A. or B.

Secondly, we interrogate the patient as to the HISTORY OF THE ILLNESS, his PERSONAL and FAMILY HISTORY, all of which require special care in nervous diseases (as mentioned in § 545).

Thirdly, we put the patient through a PHYSICAL EXAMINATION.   This has been dealt with systematically in Part B., but for a **routine examination** in ordinary cases it is sometimes convenient to adopt a regional method, beginning at the head and proceeding downwards.

(*a*) THE HEAD.—Inquire as to intelligence, sleep, pain, " attacks," or head sensations of any kind.

Eyes—Examine for visual defects, abnormality of pupils, squint, ptosis, nystagmus, changes in the fundus oculi.

Face—Notice any defect of speech, tremor of the lips, or immobility.   Test the muscles by such directions as : " Show me your teeth," " Whistle," " Screw up your eyes," " Put out your tongue."

Test the *auditory* and other *cranial nerves*, as may be necessary, in numerical order.

(*b*) UPPER LIMBS.—Examine the state of the muscles, and compare the grasp of the two sides.

Elicit any *tremor* or involuntary movement by asking the patient to extend the hands and fingers ; and direct him to touch the tip of his nose with his forefinger, and to repeat the performance with eyes closed (ataxy).

Test the supinator and triceps *reflexes*.

(*c*) LOWER LIMBS.—Examine the muscles for paralyses, rigidity, flaccidity or wasting.

Walking—Notice any peculiarity in the attitude or the *gait* ; can the patient stand with heels and toes together and eyes closed (Romberg's test) ?

Examine the *knee-jerks*, test for *ankle-clonus.*

(*d*) Test for abnormalities of CUTANEOUS SENSIBILITY (touch, pain, and temperature).

Inquire as to state of the SPHINCTERS.

Remember, once more, that there are two steps in the diagnosis of diseases of the nervous system, which, in order, are –first, the **localisation**, and secondly, the diagnoses of the **nature** of the disease.

If the symptoms point to some **generalised neurosis**, turn first to Group I (below).

If to loss of **consciousness** or **mental defect** (Group II) .. .. § 562
If there is definite **pyrexia** (Group III) .. .. § 582
If the symptoms relate to the **muscular system** (Group IV)—
    Paralysis.. .. .. § 588
    Inco-ordination or defect of gait .. .. § 613
    Rigidity .. .. § 618
    Tremor .. . . § 626
    Convulsions .. . . § 634
    Muscular atrophy .. . . § 637
If to some **sensory** or **painful** disorder (Group V) . . . § 641
If to the **special senses** or cranial nerves (Group VI) . . .. § 645
If there is some deformity of the **skull** (Group VII) .. .. § 664

GROUP I. GENERALISED NEUROSES

The word "neurosis" connotes a functional disorder of the nervous system, and by generalised neuroses are meant those which present generalised symptoms. The symptoms presented in this group of disorders are widespread and manifold, but nine-tenths of the patients *complain of nervousness*. The remarkable resemblance in their clinical features of chronic alcoholism, morphinism, and toxic neurasthenia to the other members of this group, suggests that many of the cases here met with may be dependent on some disorder of the blood.

    I. Neurasthenia.   IV. Morphinism and other drug habits.
    II. Hysteria.   V. Hypochondriasis.
    III. Alcoholism.   VI. Collapse.

§ 556. Neurasthenia is an irritable weakness of the nervous system, which may arise from a great variety of causes, and may result in many and various symptoms of nervous, mental, and bodily inefficiency.

The *symptoms* are essentially subjective. The patient may come for many different reasons : (1) A feeling of "weakness and nervousness" is one of the most usual complaints, the patient is easily tired, easily startled, easily upset. The physical debility is sometimes less marked than the mental, but it is often severe, and may even confine the patient to bed. There may be some anæmia and loss of weight. There are no physical signs unless the occasional presence of exaggerated knee-jerks, shrinkage of the fields of vision, or dilatation of the pupils may be so considered. It is convenient to describe a cerebral, cerebro-spinal, and spinal type, according to the prevailing symptoms. (2) Cerebral symptoms are always present and generally predominate. Everything the patient has to do is a trouble to him, and a source of worry, and sometimes the simplest mental work, such as adding up a column of figures, is impossible. The sleep is disturbed by dreams, or there is insomnia, or "startings" in the sleep. All the

special senses are easily tired, and sometimes the patient is quite unable to read. The pupils are usually dilated, and reaction to light and accommodation sluggish. Memory and the power of concentration may be defective. Some patients are *irritable*, egotistical, and exacting; others *gloomy* and melancholic, constantly on the grumble. (3) Spinal and musculo-sensory symptoms are also present in greater or less degree— restlessness and jerking of the limbs, weariness on the least exertion, vague pains in the back and limbs, generalised tenderness or a hypersensitive condition of the whole body, neuralgic pains and tender spots. Very often there is fine muscular tremor, but never localised paralysis or hemianaesthesia, as in hysteria. Spermatorrhœa, nocturnal emissions, or the discharge of glairy fluid at the stool, and sexual disability are common. (4) There are generally symptoms which may aptly be attributed to disturbance of the vaso-motor system—*e.g.*, long-drawn sighs, palpitation, attacks of flushing, followed by shivering, a sensation of "pins and needles" in the limbs, cold hands and feet. The patient may have a dazed feeling, as if the external world were unreal, or he may have attacks of throbbing and flushing with a sense of terror of impending death. Giddiness and faint feelings are also met with. It is in the presence of these sympathetic symptoms that neurasthenia overlaps hysteria. (5) According to some authors, gastric symptoms form an essential part of the symptoms of neurasthenia, and this is often, but not always, true. In a great many cases gastro-intestinal disorder is the cause of the trouble, and in others the asthenia of the nervous system has given rise to asthenia of the stomach (gastric myasthenia).

SHELL SHOCK.—The violent concussion of the air occasioned by the explosion of a shell is communicated to the cerebro-spinal fluid (which acts as a water-jacket to the brain and spinal cord) and causes a violent commotion of the central neurons, so great, it may be, as to cause instant death from paralysis of the medulla. Short of this it gives rise to loss of consciousness, and after this is regained to defects of memory, perception (hearing, sight, etc.) and speech, and to disturbances in the emotional sphere.

In most cases of so-called shell shock there has been no violent commotion of the nervous system : the condition is essentially due to psychic causes—the struggle between sense of duty and fear leads to temporary amnesia of some terrifying event which is thrust from the conscious mind by a process of dissociation. Recovery of the lost memory by hypnosis or otherwise, is an important factor in the treatment of these cases.

Shell shock may, as Mott has pointed out, be complicated by the effects of noxious gases. The soldier, while lying partially buried and unconscious, or at any rate helpless, may be exposed to various noxious gases, generated by shells or mines, especially CO, or oxides of nitrogen, both of which are poisonous by reason of their deoxygenating effect upon the blood. Other poisonous gases from shells may produce most injurious and even fatal results.

The evil effects of shell shock are heightened by the conditions under which the soldier exists. "Living in trenches or dug-outs, exposed to wet, cold, and often . . . to hunger and thirst, dazed and almost stunned by the unceasing din of the guns, disgusted by foul stenches, by the rats and insect tortures . . . combined with frequent grim and gruesome spectacles of comrades suddenly struck down, mangled, wounded, or dead, the memories of which are constantly recurring and exciting a

dread of impending death or of being blown up by a mine and buried alive, together constitute experiences so depressing . . . that a time must come when even the strongest will succumb, and a shell bursting near may produce a sudden loss of consciousness, not by concussion or commotion, but by acting as the last straw on an utterly exhausted nervous system " (Mott). It goes without saying that those who are congenitally neurasthenic offer less resistance to shell shock than others.

Severe shell shock may cause loss of memory not only for subsequent events, but for the whole past life—the mind may be a complete blank. The patient may suffer from terrifying auditory and visual hallucinations bearing on his actual experiences, and may go through the pantomime of fighting with bomb, bayonet, or rifle, so that he may even need to be confined to a padded room.

Mott points out that the principal objective signs and subjective symptoms of shell shock are largely those produced by fear—*e.g.*, the paralyses, tremors, the perspirations, palpitations, the cold blue hands, and mutism. Many of these features are met with in Graves' disease, and it has been suggested that the symptoms of shell shock are largely induced through the intermediary of disturbed thyroid function. It is possible that other endocrine glands are also disturbed, *e.g.*, the adrenal.

All sorts of defects of speech are met with—absolute mutism, inability to speak except in a whisper, stammering, etc. Visual defects are much less common. Every variety of hysterical manifestation may occur—paralyses, tonic spasms (common) and anæsthesias among the rest.

Treatment.—Among the most troublesome symptoms are insomnia and terrifying dreams. For these the plan recommended by Mott is the best. Place the patient for three-quarters of an hour or longer in a warm bath maintained by a special device at the blood temperature. Special attention should be paid to the condition of the alimentary tract. Headache may be relieved by an icebag applied to the head, and by such drugs as aspirin and phenacetin. Hypersensitive and restless patients should be kept in separate rooms from which noise is as far as possible shut out.

As the patient improves, mental hygiene becomes important. Above all, he requires encouragement and diversion by means of games, concerts, and the like. Gymnastic exercises, skipping, football are also helpful. As soon as he is able he should try his hand at some light occupation, especially some form of manual labour.

It must not be forgotten that recovery may be retarded by the patient's desire, subconscious perhaps, to remain an invalid, so that he may not have to rejoin the army, or with the object of securing a substantial pension.

PSYCHASTHENIA, has been described separately by Janet. It frequently complicates neurasthenia, but it may occur apart from it. The condition appears in individuals who come of a neurotic stock. They suffer from indecision and lack of will power, and dominant ideas take possession of them from time to time. These ideas may be repulsive to them, yet they are powerless to dispel them. At the same time they recognise that they are groundless, for their reasoning power is intact : they do not have delusions or hallucinations. Anxiety and morbid dreads form a prominent feature, sometimes indefinite—the fear of some unknown evil—sometimes definite, such as agarophobia (fear of open spaces), claustrophobia (fear of closed spaces—of tunnels, of being shut up in a room, etc.), monophobia (fear of being alone), anthropophobia (dread of society or of people). Sometimes the dreads take the form of paroxysmal panics or terrors, which pass off as suddenly as they come, and I have seen several such cases drift into melancholia and dementia.[1] There is no doubt that the miseries

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[1] " Clinical Lectures on Neurasthenia," 4th edition, J. H. Glaisher and Co., London, 1908.

of neurasthenia and of psychasthenia are very real, and not a few patients, particularly those of the gloomy type—who go wearily on with their work, saying but little of what they feel, who have probably never actually threatened suicide—seek relief by terminating their lives. Other patients drift into a chronic and incurable hypochondriasis.

In the *Diagnosis* the very vagueness of the symptoms is an aid. Neurasthenia is certainly not the same disease as *hysteria* (see table), nor is neurasthenia a new name for hysteria, as some suppose, though the two conditions overlap in some respects. Hysterical phenomena are paroxysmal and recurrent, with intervals of health; in neurasthenia the symptoms are more or less continuous and occur almost equally in either sex.

Prognosis.—Neurasthenia is essentially a chronic disease, and leads to a great deal of misery, but is never fatal, except by suicide. Some hold that the condition is incurable. The author, however, believes it to be largely curable, provided a careful investigation be made, leading to the discovery of the cause or causes in operation. It may run on for years, especially if, as is so frequently the case, dyspepsia, colitis, or some other chronic cause is in operation. Its prognosis depends chiefly on (i.) its previous duration, and (ii.) the removability of the cause, and (iii.) the age of the patient, being most favourable in young persons.

Causation.—The causes of neurasthenia may be classified into four groups, compound causes being very ████████ in operation : (1) *Toxæmic* causes, which include dyspepsia, col████████ other gastro-intestinal disorders, such as chronic constipation, dilatation or kinking of the colon, bad teeth or pyorrhœa alveolaris, and various other infective foci. Abuse of alcohol, morphia, or cocaine, and Graves' disease. (2) *Malnutrition*, such as is produced by post-influenzal (a specially potent cause) and other post-febrile and debilitating conditions, and deficient or unwholesome food. (3) *Fatigue* and over-functioning, e.g., prolonged overwork, worry, anxiety, deficient or defective sleep, prolonged pain and excessive sexual indulgence. (4) *Emotional* and *traumatic* causes—grief, shock (mental or bodily), railway and other accidents, in which the neurasthenia need not supervene until a few weeks after the shock. Injury may produce either "traumatic neurasthenia" or "railway spine" (see Paraplegia). The now all too familiar "shell shock" is a potent cause of neurasthenia. My statistics (*loc. cit.*) show that some underlying gastro-intestinal cause is present in 75 per cent. of the cases. A hereditary predisposition plays a part in the etiology—though not, in my belief, as prominent a one as some hold. In regard to age, no age is exempt, but neurasthenia is commoner in young and middle-aged adults than in advanced age or in children. Sixty-one per cent. of the cases under my care were males. A sedentary indoor life, the unhealthy atmosphere of town life, and the rush and strain of modern civilisation favour the occurrence of the disease.

The *Treatment* resolves itself briefly into : (1) The alleviation of the distressing symptoms as far as may be. Bromides are particularly useful in this respect : morphia and other sedatives require great care. Alcohol

TABLE OF DIAGNOSIS.

	Neurasthenia.	Hysteria	Hypochondriasis.
Sex.	Both sexes almost equally.	Female sex almost exclusively	Males chiefly affected.
Age.	Any age—young men slightly predisposed.	Definite manifestations of some kind appear for the first time practically always before twenty-five, generally between fifteen and twenty.	Rare under thirty; predisposition from thirty to fifty
Causes.	Produced by overwork, gastro-intestinal and other causes of toxæmia, defective metabolism; occasionally shock	Subjects of the hysterical diathesis are liable to hysterical manifestations throughout life, the determining cause always being an emotional upset.	Solitary, sedentary life, prolonged gastro intestinal trouble
Onset.	Starts gradually and runs a fairly even course.	Onset sudden, generally with an attack of some kind; all manifestations vary from hour to hour and day to day.	Starts gradually, and runs an even and intractable course of indefinite duration
Mind.	Mental exhaustion and inability to think, study, or do work of any kind; memory deficient; intellect clouded for business, always tired; temper irritable; depression, sometimes suicidal.	Wayward, impulsive, and emotional; fond of gaiety and amusement; usually joyous, but laughter and tears alternate with great rapidity; memory and intellect sometimes brilliant; no tendency to suicide	Introspective habit, mind concentrated on bodily functions, habitual sadness, no taste for amusement; patient tries an endless succession of remedies and doctors, always striving for a cure.
Bodily symptoms	Chronic weakness and nervousness; vague sensations about the head; convulsions never; gastro-intestinal trouble of some kind in 75 per cent.	Symptoms paroxysmal; seizures of different kinds frequent; flush readily; attacks of globus and syncope frequent; convulsive attacks in nearly half the cases.	No seizures of any kind, runs an even course
Result.	May last a long time, but by appropriate measures a certain proportion are CURABLE.	Active manifestations disappear suddenly and unexpectedly; very apt to recur, and therefore only TEMPORARILY CURABLE.	Once established hypochondriasis is impossible to eradicate, progressive and INCURABLE.

and tobacco should be avoided. (2) The removal of, or compensation for, the above causes. Dyspepsia is in evidence in nearly all out-patient cases, and it is surprising how efficacious an alkaline gentian mixture is in these. Aperients and intestinal antiseptics are valuable. Regular outdoor exercise is useful. (3) Nerve tonics with hygienic and educational measures conducted with sympathy and encouragement will do much for those in whom hereditary predisposition is strong. Among nerve tonics strychnine is very useful, and arsenic, phosphorus, phosphates, glycerophosphates, malt, and cod-liver oil may be tried. Turkish and warm baths allay irritability. Cold baths and judicious hydrotherapy, various forms of electricity, and a regulated diet have all done service in these cases. (4) Complete physiological rest of the nervous system is of the greatest value. but I do not recommend Weir Mitchell treatment—at least, in its

complete form. Sea voyages are beneficial. (5) In the most intractable cases operations on the colon, as suggested by Sir A. Lane, may be considered (6) A knowledge of psychotherapeutics is an essential addition to the physician's armamentarium in the treatment, especially of the emotional varieties of this disease and of psychasthenia.

§ 557. **Hysteria** is a word derived from ὅστερον, the womb, in the mistaken belief, prevalent in the Middle Ages, that the disorder, which is almost confined to the female sex, arose within that organ. It may provisionally be defined as a condition of instability of all the emotional, vaso-motor, and all the reflex nervous functions, with a tendency to the development from time to time *throughout life* of many different forms of nervous seizure, and of various motor and sensory disorders closely resembling organic diseases of the nervous system, never leading to a fatal issue.[1] A prominent feature of the disease is the tendency to *disintegration of the mental personality*, as pointed out by Janet.

Symptoms The hysterical disposition is essentially one of unstable equilibrium. These patients are easily aroused to violent expressions of feeling, hasty judgment, impulsive actions, and passionate exhibitions of various kinds. They are highly suggestible. There is hypersensitiveness to all forms of pain, and a tendency to neuralgia, the favourite seats for which are just below the left breast, or on one side of the head (clavus). The hysterical diathesis may also be suspected by the presence or a history of any of the following " hysterical stigmata." [2] (1) *Flushings* of the face and other parts with or without provocation. Sudden pallor and other vaso-motor phenomena are also very frequent in hysterical subjects.[3] (2) *Nervous attacks* of some kind always occur sooner or later —" hysterics " (crying and laughing), nervous faints, etc. They are especially common at the catamenial period, or after some " contrariment " emotional disturbance Hysterical fits have to be distinguished from epileptic fits. The patient rarely hurts herself in falling, the movements are purposive rather than inco-ordinate, though the lips may be bitten the tongue is not, she does not pass water during the fit, the face is not livid, the eyes may be tightly closed, the fit is not followed by coma. It is often possible to reinduce a hysterical fit by suggestion, especially during hypnosis. The Hysterical fit may follow upon a true epileptic fit, especially of the petit mal variety. *Hystero-epilepsy* (Charcot's *grande hystérie*) is rarely seen in this country. In *hysterical catalepsy* the patient becomes stiff, motionless and speechless. In the hysterical trance the patient sinks into an apparent sleep which may last from hours to weeks. (3) "*Globus*" is a sensation as of a ball in the throat, or a sense of choking or suffocation. Flatulence is a frequent accompaniment of globus, and a severe attack of

[1] " Lectures on Hysteria and Allied Vaso-Motor Disorders," Glaisher and Co., London, 1909.
[2] A stigma etymologically signifies an abiding mark or sign by which something may be recognised.
[3] See a clinical lecture on the " Skin Symptoms of Hysteria," the *Lancet*, January 30, 1904 ; and " Lectures on Hysteria."

globus with prostration is often terminated by copious windy eructations. (4) "*Ovarie*," or the hysterical ovarian phenomenon, consists of a tenderness on pressure on either inguinal region, which produces an indescribable feeling rising up towards the heart and throat. Pressure in this, the "ovarian" region, may determine some kind of attack. This phenomenon is in no way dependent on the ovary, but as I have elsewhere shown,[1] is specially related to the ilio-hypogastric and ilio-inguinal nerves. Similar hyperæsthetic or hysterogenic zones may exist elsewhere. (5) *Patches of anæsthesia or hyperæsthesia* may exist almost unknown to the patient. The anæsthesia may occupy one half of the body and involve the special senses on that side. *It is always the result of suggestion.*

Hysterical disorders may affect any part of the body, closely simulating organic disease. These are described under their appropriate sections; a brief summary only is given here. (1) Disorders of motion paralysis of the voluntary muscles—hemiplegia, monoplegia, rarely if ever paralysis of the face; rigidities; tremors and convulsions of one or several limbs, or of the whole body. (2) The involuntary muscles may also be affected by paralysis or spasm—adductor spasm or paralysis (aphonia) of the vocal cords, dysphagia, hiccough, cough, dyspnœa, borborygmi, phantom tumour (from paralysis and ballooning of a portion of the intestine), vomiting without nausea. (3) Anæsthesia may affect one limb or half of the body; hyperæsthesia, especially of the spine, and various neuralgiæ are common (4) Joint affections, with pain and stiffness, may occur. (5) Hyperpyrexia as indicated by the thermometer is the result of tampering with the instrument. (6) The special senses may be affected—amaurosis, retraction of the field of vision, deafness, disturbance of taste and smell. (7) There may be mental disorder—trance, catalepsy, hallucinations and delusions —especially after convulsive attacks. (8) Finally, the patient may exhibit an alternating personality.

The *Diagnosis* of hysteria from neurasthenia has been considered in § 556. The salient features of hysteria are its limitation practically to the female sex, the *paroxysmal occurrence* of all its symptoms, and a previous history of similar symptoms. The diagnosis of the numerous hysterical phenomena is dealt with under the various disorders which they most resemble.

Prognosis.—The hysterical diathesis persists throughout life, modified from time to time by the state of the health and the surroundings. The disease never terminates fatally of itself, but it often renders the patient's life a misery to herself and those about her.

Etiology.—Hysteria is practically confined to the female sex; in the male sex it only occurs to the extent of about 2 per cent. Heredity is a potent factor, and can be traced in at least 75 or 80 per cent.; the influence is transmitted mainly through the mother. In many cases there is a family history of one or other of the diseases dealt with in this group (Group I). Faulty education, the spoiling of children, a life of self-

[1] The *Lancet*, July 20, 1901, p. 122.

indulgence, or any mode of existence which leads to undue introspection, or a diminution of the control normally exercised by the will, undoubtedly foster the diathesis, as also do faulty hygienic conditions. The favourite ages for the occurrence of hysterical phenomena are soon after the evolution and at the involution of a woman's sexual life. Typical hysteria is, however, met with in children and even in old people, though rarely. The determining cause of all hysterical manifestations is some emotional shock, trivial or severe. No anatomical or histological lesions have yet been discovered.

Of recent years the psychic origin of hysterical disorders has been receiving much attention. Janet defines hysteria as "a form of mental depression characterised by a shrinkage of the field of personal consciousness, and a tendency to the dissociation of the systems of ideas and feelings that constitute personality." Sudden emotion is the usual cause of this disintegration of the personality. Freud believes that hysteria may originate in a painful reminiscence which has been "repressed" or in a repressed wish. The repressed material is supposed to lead an *active* existence in the *unconscious* mental life of the patient and to express itself symbolically in the form of mental and bodily symptoms. Such repressed memories can be recalled by psycho-analysis. Once the buried reminiscence has been restored to the consciousness, and the mental attitude towards it has been altered by re-education, the physical manifestation disappears. Babinski regards hysteria as made up of symptoms which are engendered by suggestion and removable by persuasion and suggestion. Persuasion relies upon logical argument, suggestion works independently of argument.

Treatment.—Educational treatment is very important, and as few mothers of hysterical girls possess the necessary combination of judgment, firmness, tact, and kindness the treatment of such should be entrusted to others. Some regular occupation and interest in life is essential. Idleness and a frivolous empty life are calculated to foster the diathesis. Matrimony thus becomes a valuable adjuvant, because it gives to a young woman occupation, interests, and responsibilities outside herself. Briquet[1] has shown conclusively that hysteria has no causal relation to ungratified sexual passions. If the manifestations of the diathesis are sufficiently pronounced, a course of treatment is indicated which comprises (1) removal from the conditions under which the disease is fostered, (2) isolation from sympathetic friends, (3) abundant feeding with milk and other easily assimilable foods, and (4) massage, which enables the patient to take and assimilate more food. These four measures constitute the Charcot or Weir Mitchell method of treatment. The treatment for the nervous attacks, when they arise, consists of the sudden application of cold water to the face, the faradic battery to the limbs, and the internal administration of asafœtida, valerian, and spirits of ether, or chloroform. A hypodermic injection of apomorphine effectually terminates hysterical convulsions and other violent seizures.

§ 558. **Hypochondriasis** is a morbid condition of the nervous system allied to neurasthenia on the one hand, and melancholia on the other. It is an introspective,

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[1] "Traité Clinique et Thérapeutique de l'Hystérie," par le Docteur Paul Briquet, p. 206. Paris, Baillière et Fils, 1859.

melancholic, or pessimistic habit of the mind, in which the patient believes, without cause, that he is the subject of one or more serious bodily disorders. He is gloomy, wrapped up in himself, but talkative; a slight pain in the stomach is certainly cancer; a slight palpitation is regarded as mortal cardiac disorder; or the testicles hang too low, and therefore he will certainly become impotent for life. Yet, in spite of all, he is not without hope, for he will spend his life taking physic and frequenting the consulting-rooms of physicians, surgeons, and quacks—where he will argue "learnedly" about his symptoms until the unhappy physician wishes he had not been born.   Nor are these patients suicidal.   On the other hand, the true melancholiac is hopeless and often suicidal.

Diagnosis.—Hypochondriasis used to be regarded as the masculine equivalent of *hysteria*, but it is a very different affection (table in § 556).   It bears a closer relation to *neurasthenia*.

Causes.—Hypochondriasis is occasionally seen in the female, about the menopause, but the patients are mostly men of middle age.   It is rarely seen before puberty, or indeed, before thirty, and generally makes its first appearance between thirty and forty.   There is often a neurotic family history, including insanity.   Digestive disorder (gastro-intestinal or hepatic) is always present, and may be looked upon as its most frequent cause—a fact of interest in connection with the marked prostration and depression which attend gastric and abdominal disorders.   Flatulence and dilatation of the stomach are common.   I am satisfied that many cases which were formerly, and are still, regarded as hypochondriasis are in reality intractable cases of neurasthenia, due to dilatation, kinking, stasis or intestinal toxæmia.

Treatment is neither easy nor satisfactory.   The dyspepsia should be treated, and a draught of ammoniated tincture of valerian, or pil. asaf. co., or some other antispasmodic, taken occasionally for the flatulence.   It gives relief, not only to the flatulence but to other symptoms.   The bowels should be carefully regulated and otherwise treated.   These means, with regular exercise, constant change, cheerful society, help to break through the vicious mental attitude; but complete recovery rarely if ever occurs (see also Treatment of Neurasthenia, § 556).

§ 559. Alcoholism or excessive indulgence in alcohol is met with clinically in three forms : (1) Acute alcoholism, (2) chronic alcoholism, a phase of which constitutes (3) delirium tremens.

Acute Alcoholism is due to an excessive quantity taken in a few hours. It gives rise to mental disturbance, muscular inco-ordination, and finally narcosis with a marked lowering of the body temperature, and a heavy alcoholic odour of the breath.   The stupor of apoplexy, uræmia, opium poisoning, etc. (§ 564), and the muttering delirium (§ 372) of pneumonia and other diseases, may be mistaken for drunkenness, a serious error which is best avoided *by keeping the patient under observation in bed* and suspending our judgment.   A recurrent craving for alcohol leading the subject to drink himself into a state of coma is known as *dipsomania.* The attack lasts a few days only.   In the intervals there is complete sobriety, or even a distaste for alcohol.   There is generally a neurotic family history.

Chronic Alcoholism is due to the persistent imbibition of moderate doses of alcohol over a long period of time.   The effects are worst when the alcohol is taken on an empty stomach or in the form of raw spirits. It acts as a poison on the nervous, muscular (voluntary and involuntary), and epithelial elements, and hinders tissue oxidation, so leading to fatty degeneration.

The *Symptoms* in the earlier phases resemble neurasthenia in many respects, with special toxic effects added later. The *neuro-muscular system* early shows signs in (i.) the unsteadiness and tremor of muscles, especially those of the hands, and in peripheral neuritis, of which alcohol is the most frequent cause. (ii.) The mental processes become slow, the memory, judgment, and will enfeebled, and later dementia or some other form of insanity may ensue. Delirium tremens (see below) supervenes from time to time, and sometimes epileptiform convulsions. The *digestive system* is generally affected. (i.) Chronic gastric catarrh, attended by characteristic morning vomiting, is always present in spirit drinkers, and gastric dilatation in beer drinkers. (ii.) Some hepatic congestion occurs in all cases, and cirrhosis (with or without fatty degeneration) is common in spirit drinkers. The *heart* dilates and undergoes fatty degeneration, and the vessels become thick and degenerated. This form of fatty heart is a common cause of sudden death. The *kidneys* become congested, enlarged, and later cirrhotic. The *facies* of the chronic toper is characteristic - redness of the cheeks and nose, with œdema of the conjunctivæ. Often, however, the complexion is quite normal, as anyone visiting a home for inebriates can convince himself.

Dipsomania is that condition in which the patient is periodically seized with an overpowering craving for alcohol. The drinking bout lasts for a few days, after which the patient may have an actual disgust for alcohol. *True dipsomania* develops spontaneously. *Pseudo-dipsomania* comes on in the chronic toper who after abstaining for a time ventures upon the fatal " first glass."

Delirium Tremens (*delirium e potu*).—Dr. Francis Hare has conclusively shown that this is due to the sudden reduction in the amount of circulating alcohol in a chronic heavy drinker, and that it can always be prevented by gradually tapering off the alcohol instead of suddenly withholding it. Such a sudden reduction may have been enforced or have resulted from vomiting. It is probable that the delirium tremens which complicates operations or acute diseases such as pneumonia is due to the reduction of the usual daily allowance of alcohol. Incoherent mutterings or ravings, characterised by hallucinations of vision (insects, spiders, or rats), accompanied by muscular tremor, intractable sleeplessness, and in bad cases two or three degrees of fever, are the leading features of the malady, which usually runs its course in two to five days.

The *Diagnosis* of chronic alcoholism is generally easy. The diagnosis of delirium tremens is referred to in § 372. Care should be taken not to overlook acute pneumonia, particularly of the apex. The *Prognosis* of delirium tremens is generally favourable if the temperature is not much elevated and the strength of the patient can be maintained.

The *Treatment* of acute alcoholism consists of the administration of an emetic, such as zinc sulphate, or apomorphine, ⅒ grain, hypodermically, and a large dose of calomel. A chronic alcoholic habit is rarely abandoned after forty, and residence in a home is advisable in all confirmed cases, to

enable the patient to regain his self-control. In other cases much may be done by careful domestic control and medical supervision. The gastric catarrh must be treated, and the uncomfortable " sinking " feelings may be much relieved by tincture of capsicum ℳ v., sod. bic. gr. x., in an ounce of peppermint or chloroform water. In acute and chronic alcoholism one of the most distressing symptoms is insomnia, and it is difficult to treat. In the acute form opium is said to be contra-indicated, and it is doubtful if chloral or other hypnotics will shorten the attack. Digitalis in large doses may be tried, but it will be found that large doses of bromides, and in some cases opium, are generally successful. For dipsomaniacs and those who are born with an enfeebled nervous system, little can be done unless they will surrender the control of their lives to others. The great value of strychnine, atropin, and cinchona in the treatment of alcoholism and morphinism has no doubt been known to several observers for some years, and I cannot speak too highly of them as regards my own experience. Strychnine and atropin sulphates (hypodermically), $\frac{1}{60}$ grain and $1\frac{1}{2}$ grain respectively, may be given with cinchona bark (by the mouth), four times daily until the throat is dry and the pupils dilated.[1] No medical man should countenance a secret method of cure. The moral influence of a well-ordered institution is a highly important factor in the success of treatment.[2]

In all cases of chronic heavy drinking the daily allowance of alcohol should be tapered off cautiously, never suddenly cut off, otherwise delirium tremens may be induced. The treatment of *delirium tremens* calls in the first place for a reliable attendant (perhaps two or three), for the patient may be violent in his attempts to escape from his horrible visions, and artificial restraint may be necessary. *Alcohol should be given freely in the early phases and gradually tapered off.* To procure sleep and maintain the strength by nourishment are the main indications. Large doses of bromide, and chloral (if the pulse is not too weak) or hyoscine, $\frac{1}{100}$ grain (0·0006), may be tried ; but, as a rule, the malady runs its course unaffected by drugs. Graduated cold baths and cold packs are often very efficacious in cases with pyrexia (§ 422).

§ 560. **Morphinism** (Synonym : **Morphia Habit, Morphinomania**) and other drug habits.—Hypodermically, morphia in small doses is a nerve stimulant as well as a hypnotic, and induces a feeling of contentment and well-being ; but in the course of twenty-four hours reaction and craving for more occur, particularly when pain is present, and by degrees the dose has to be increased until in the course of a few months twenty to one hundred times the normal dose is necessary to produce a feeling of satisfaction, and can be easily tolerated. The only *signs* by which the *morphine habitués* can be detected are contracted pupils, pallor of the face, and the frequency with which they withdraw to satisfy their craving—a difference being observed in their depression before and their gaiety and brightness afterwards.

If such a patient is suddenly deprived of the drug, the following *symptoms* (which

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[1] Mr. S. B. Fenn, *Brit. Med. Journ.*, 1904, vol. i., p. 1008 ; and Dr. C. A. McBride, *Brit. Med. Journ.*, 1904, vol. i., p. 1006.

[2] " Alcoholism and its Treatment," Dr. Francis Hare.

I have been accustomed to call "amorphinism") set in. The pulse, which was previously normal, becomes rapid and of low tension, and the patient prostrate, suffering agonies from tingling in the limbs, sweatings, sneezings, lachrymation, diarrhœa, vomiting, uncontrollable restlessness, faintings, sinkings in the pit of the stomach, extreme wakefulness, and a host of horrible and indescribable somatic sensations resembling extreme neurasthenia.

Consequences of the morphia habit. Enormous doses may be taken by gradual increase ; one of the largest in my experience was 25 grains (1·6) a day, reached after a habit of only two years. At first the patient is always gay, and has great capacity for mental and bodily endurance. But if the habit be continued, the character gradually becomes altered. The patient alienates his friends by his tempers and unreliability ; and, one by one, truth, reverence, and honesty disappear. If there be difficulty in procuring the drug, great craftiness is exhibited, and cases are known of women previously of the highest character selling their virtue and their husband's honour to procure it. In course of time the mental powers gradually deteriorate, and suicide is not infrequent in those who desire, but are unable, to rid themselves of the thraldom. The body also suffers, and the patients become pale and emaciated. They get careless in the use of their syringe, multiple abscesses form, and death may result from septicæmia. Fatty degeneration of the viscera ensues, especially of the heart. My own belief is that some of the cases of sudden death attributed to overdose of morphia are due to this condition.

Prognosis.—That the habit shortens life is certain, though it may continue for many years. The danger of sudden death from the causes just explained, or from an overdose, is considerable. The curability of a case of morphinism depends on three points : The age of the patient, the duration of the habit, and the curability of the painful affection for which it was first contracted. The actual quantity per diem which has been reached is of small account. A habit of 4 grains (0·25) daily of ten years' duration was more difficult to cure than one of 22 grains (1·4) of two years' duration. If carcinoma or some other cause of an incurable and recurrent pain be present, and especially if the patient be aged, the tendency to relapse is great, and it may be impossible to ease the pain in any other way.

Treatment.—(a) To break the habit the patient must place himself under the absolute control and guidance of a physician in whom he has confidence. Three methods are advocated—sudden cessation, gradual reduction, and gradual reduction combined with the substitution of solid opium or morphia by mouth or rectum. The first of these should never be employed unless time is an object, for the suffering is very great, and if the habit be of long duration, not without danger. In the third, which is advocated by Dr. Oscar Jennings, of Paris, a definite scheme is written out by the physician, in which about double the quantity of opium, by mouth or rectum, is allowed to replace the gradual reduction in the morphia. Afterwards, when all hypodermics have ceased, the opium internally can be gradually reduced without much difficulty. The second method, gradual reduction, is the one which I have found most successful. Cut down at once to one-half, then gradually reduce. There should be no difficulty in reducing, by ¼ grain (0·018) a day, down to 2 grains (0·13), after which the reduction should continue in quantities less and less in a geometrical ratio by the daily addition of sterilised water to the stock-bottle. The greatest difficulty is with the last few doses, which patients may cling to for weeks or months.

(b) Treatment of the symptoms of amorphinism. The prostration and somatic sensations may be partially relieved by moderate doses of alcohol, ammonia, and other stimulants, or by strychnine, 4 minims (0·2) hypodermically, and atropin, as recommended in § 559. We may be compelled to allow the patient some morphia. Digitalis and other drugs which aid the heart are useful. For the vomiting and diarrhœa bismuth is best, but the diarrhœa should not be checked too much. Dionin relieved the restlessness and craving in one case under my observation. For the sleeplessness, chloral and other hypnotics and analgesics may be tried, but are of little use. After recovery there is a tendency : (1) towards relapse, and therefore

the patient should be kept under observation ; and (2) towards alcoholism, and there-fore great care is required in the administration of stimulants. Another method of treatment is by the administration of hyoscine hydrobromate hypodermically gr. $\frac{1}{100}$, and every hour after this gr. $\frac{1}{500}$, for 24–48 hours, until intoxication supervenes. Thereafter mild intoxication is maintained for another 24–48 hours, when the drug is stopped.

A heroin habit can cause as serious symptoms as a morphia habit.

The cocaine habit leads to many of the troubles of the morphia habit, but there is a greater tendency to mental symptoms and mania. Morphia and cocaine is a frequent combination, and in such cases the cocaine may, with comparative ease, be first withdrawn. Then the morphia reduction may be proceeded with as above described.

The chloral habit is not so common nowadays as the preceding. It gives rise to gastro-intestinal disturbance, lowered nutrition, pains, skin eruptions, depression, and irritability, palpitation, and cardiac weakness. Sudden death may occur from slight increase of the dose.

Sulphonal, phenacetin, antipyrin, and other tar products do not so readily engender a craving, but when habitually used the patient cannot do without them, and in course of time symptoms similar to those of chloral hydrate arise.

§ 561. Collapse (or Shock) is a condition of extreme prostration. An attempt is sometimes made to distinguish shock and collapse, but the two are clinically identical. The term "shock" is applied to that condition which follows any sudden mental or physical injury ; the term "collapse" when it supervenes on some less sudden cause, such as cholera. In the traumatic form there is a rapid draining of blood plasma from the capillaries. There is paralysis, or more properly paresis, of all the muscular tissues of the body, voluntary and involuntary (muscles of the limbs. of respiration, of the heart and arteries). The *Symptoms* may be arranged under the following headings : (1) The skin (especially of the extremities) is pale, cold, and clammy ; the surface temperature is 2° F. or more under normal ; the pupils are dilated, and react slowly to light. (2) The circulation and respiration are very feeble, the pulse being rapid and scarcely perceptible. (3) Loss of voluntary movement (sometimes rest-lessness and, in cases of profuse hæmorrhage, convulsions). There is apathy, but the intellect is clear. The urine and other secretions are diminished or suppressed. The patient may die, or may pass into a reaction stage, with slight pyrexia.

Diagnosis.—In *coma* the mind is completely obscured, and the respiration laboured and stertorous. Save for the functions of organic life, all is in abeyance. In *syncope* consciousness is generally lost, but the condition is more transient.

The *Causes* of collapse may be divided into those of sudden and those of gradual onset. When the condition is of sudden onset after injury or emotion, it is usually described as shock.

(a) Of *sudden* onset : (1) An overdose of chloroform or ether. (2) Surgical operations or severe injury. A vigorous man suffers more from operation than an old man or one who has been in bed for some time previously. Blows on the abdomen or extensive burns are always attended by more or less collapse. A relatively slight injury to a very sensitive

part, such as the testicle, may produce collapse.   (3) Severe and sudden emotions (terror, grief, or war shock), or acute pain coming on suddenly, such as biliary or renal colic.   (4) Poisoning by the narcotic irritants (oxalic acid, carbolic acid, phosphorus, etc.) and by the asthenic poisons (hydrocyanic acid, aconite, digitalis, tobacco, veratria) ; ptomaine poisoning from tinned meats, etc.   In cases of anaphylaxis (§ 418) collapse may occur. (5) Profuse hæmorrhage or diarrhœa, as in post-partum hæmorrhage and cholera.   (6) Intestinal obstruction.   (7) Perforation of some part of the alimentary canal, with extravasation of its contents into the peritoneum. (8) Rupture of an abdominal cyst or of an abdominal or thoracic organ. (9) Pulmonary or other embolism.   (10) Heat exhaustion after exposure to a very hot sun.

(b) Of *gradual* onset : (1) Privation and exposure combined.   (2) Profuse diarrhœa, such as usually terminates amyloid disease.   (3) Peritonitis and other abdominal inflammations.   (4) The asthenic types of fever, such as may attend enteric and yellow fever.   (5) The termination of many diseases described in the chapter on Debility.

When a patient is found in a state of collapse or shock, the physician has to diagnose the *cause* of the condition.   After applying restoratives he should inquire : first, whether there is a history of injury or emotional disturbance, hæmorrhage, etc. ; secondly, if the patient was in good health up to the time of onset of the condition, so as to exclude group (b) ; thirdly, what food the patient has recently taken, remembering the possibility of poison.   Finally, he should examine all the viscera, especially the heart and abdominal organs, beginning at the part which is or has been the seat of pain.

The immediate *Treatment* consists in applying warmth to the body by means of hot bottles and warm blankets.   The head should be lowered, the feet raised, especially in cases following external hæmorrhage.   Stimulants (alcohol or ammonia) may be given by the mouth if the patient can take them.   Hypodermics of ether (℥ 20 to 60 (1·3–4) every half-hour), or liq. strych. (℥ 10 (0·6)), or brandy should be given.   Normal saline or isotonic glucose solution may be administered subcutaneously, intravenously, or per rectum (§ 436), and a subcutaneous injection of pituitary extract (1 c.c.) every four hours.

GROUP II. MENTAL SYMPTOMS

Disorders of consciousness form a very large group.   There are not many serious disorders of the nervous system in which mental symptoms do not appear sooner or later.   These are generally omitted from textbooks on medicine, but the scheme of this work would not be complete without a brief epitome of the more important.

It will be found convenient to deal with mental symptoms under four headings :

(a) Sudden interruptions of consciousness—*slight and transient*, such as petit mal ; or *severe and prolonged*, such as coma ..   §§ 562 and 564

(β) Partial mental and " one faculty " defects, such as speech defects,
    or loss of memory   ..   ..   § 567
(γ) Acute perversions of the mind, such as delirium and mania ..   ..   § 571
(δ) Chronic perversions of the mind in adults, adolescents, and children, such
    'as melancholia, dementia, and various special types   ..   § 572 *et seq.*

The patient complains of sudden transient **attacks of unconsciousness.**
The case is probably one of SYNCOPE or EPILEPSY MINOR.

§ 562. **Sudden,** usually **brief and transient unconsciousness.** --The patient
comes, perhaps, complaining of " attacks," " sensations," " faints,"
" dizziness in the head," or " interruptions of thought." You have only
the patient's account to guide you, and it may be a little difficult to
diagnose the condition, but it is probably SYNCOPE, EPILEPSY MINOR,
ARTERIAL (or senile) VERTIGO, OR SOME FORM OF AURAL VERTIGO.

It is necessary to ascertain first whether the patient was really uncon-
scious (Did he know who was beside him, or hear when they spoke to
him, and did he fall down ?) ; secondly, the age and sex of the patient- -
epilepsy appears for the first time between ten and thirty, hysterical
faints are almost always confined to young females ; thirdly, the history
of previous attacks.

I. **Syncope** is a loss of consciousness more or less complete, generally
of short duration, due to cardio-vascular failure. It does not generally
last longer than a few minutes, though the duration and intensity vary
considerably. Before the attack the patient is pale, sometimes a useful
warning. The process of going off is accompanied by a disagreeable
" swimming in the head," which has some resemblance to vertigo, or by
an indescribable sinking feeling in the region of the stomach. The process
of recovery is more gradual than in petit mal. The diagnosis of cardiac
syncope from petit mal, and their causes and treatment, have been given
in tabular form, § 26.

II. **Epilepsy Minor** (Synonyms : Petit Mal, Epileptic Vertigo).- There,
are two varieties of idiopathic epilepsy, E. major (§ 635), consisting of
unconsciousness with convulsions, and E. minor (petit mal), unconscious-
ness without convulsions. They sometimes alternate in the same indi-
vidual. There are many degrees, shading from one to the other, and
few dispute the identity of the two maladies. Only one circumstance
suggests a contrary view—namely, that amyl nitrite will often stop an
attack of major, but increases the severity of an attack of minor, epilepsy.
Epilepsy minor may be defined as a momentary or brief loss of con-
sciousness, preceded—in about half the cases—by an aura or warning,
unattended by convulsions, and often without falling, not followed by the
stage of stupor, the whole lasting rarely more than half a minute to a
minute. In the attacks now under consideration, it may be that the
patient merely pauses in a conversation, or there is only a vacant look, a
fixity of gaze, dilated pupils, or momentary pallor of the face, which none
but a close observer would notice. More usually, however, the patient
—who generally refers to these attacks as " sensations "—feels giddy

and loses his equilibrium for a few moments. Though usually lost, consciousness is *not necessarily lost in petit mal* ; there may, *e.g.*, be a twitching of the face and pallor without any interruption of consciousness. The absence of any apparent rotation of objects helps us to distinguish epilepsy minor from vertigo ; and its appearance always for the first time in the earlier decades of life distinguishes it from " senile syncope." The attack may be followed by a period of automatism (post-epileptic automatism) in which the patient may perform a number of irresponsible—even criminal—acts of which he has no recollection when he recovers his normal mental state. These cases are of considerable medico-legal interest. Petit mal is more likely to result in dementia than grand mal. The *Treatment* is on the same lines as that of epilepsy major (§ 635).

MASKED EPILEPSY is a still more incomplete form of epileptic attack, in which the patient, though unconscious, continues to perform automatically various acts during the seizure. Thus, a shoemaker under my care would continue his sewing ; and Trousseau mentions a skilled violinist who continued to play with precision. He also mentioned a judge who used to leave the Bench during an attack of masked epilepsy, micturate in a corner of his robing-room, and return to the Bench again without a return of consciousness. The criminal records show that homicide may be performed during these attacks.

III. **Senile Syncope ; Senile or Arterial Vertigo ; Apoplectiform Attacks.** Under these terms may be included attacks of unconsciousness, varying in degree from a transient interruption of thought to total loss of consciousness. Those who have much to do with old people are aware that brief lapses of consciousness are extremely frequent among them. Sometimes they amount to nothing more than a momentary confusion of thought. They were variously described by my old patients in the Paddington Workhouse as " dizziness " or " giddy faints." They often give rise to a momentary reeling if the patient happens to be standing or walking. But sometimes there is a definite unconsciousness, lasting one or two minutes, or longer, in which he falls unless he leans against or catches hold of something. Every gradation is met with between these minor attacks and a severe syncopal or apoplectic seizure. They are generally associated with arterial hypermyotrophy [1] or some form of arterial degeneration, especially when this is combined with cardiac failure. They are doubtless due to temporary failure of the cerebral circulation. In a few instances of the severer form of attack I found after death minute softenings or hæmorrhages of various dates, sometimes in considerable numbers, with which the attacks were undoubtedly connected.

IV. **Aural Vertigo** (§§ 544 and 657).—Patients often speak of attacks of aural vertigo as " faints," though they are not usually attended by unconsciousness. It is not, however, recognised that severe attacks of aural vertigo may occasionally be attended by complete loss of consciousness of some duration, and that such attacks may be connected with middle-ear catarrh as well as lesions of the inner ear.

§ 563. V. **Ménière's Disease** (or Labyrinthine Vertigo) is due to hæmorrhage into the vestibule or semi-circular canals. There is sudden loss of consciousness, followed by recurrent attacks of paroxysmal vertigo, associated with nerve deafness. This is the classical disease. The term is sometimes applied to any form of vertigo associated with deafness, but this does not tally with Ménière's original description,[2] and the name is better exclusively reserved for vertiginous attacks attended by loss of consciousness, and associated with deafness.

[1] Trans. Path. Soc. Lond., 1904.
[2] It seems that all Ménière's original cases came on in an apoplectiform manner. *Gazette Médicale*, 1861.

There are four classical symptoms : (i.) The vertigo always occurs in paroxysms in which, after the first attack, consciousness is retained, though the patient may fall, owing to the disturbance of equilibrium. Irritation of the left semi-circular canals usually produces a sense of rotation from left to right; destruction the reverse. (ii.) The attacks are commonly attended by nausea, or even vomiting. (iii.) There is deafness in the great majority of cases, on one or both sides, the patient being unable to hear a tuning-fork placed on the teeth or the head (perosseous—*i.e.*, nerve deafness) ; and (iv.) tinnitus, or buzzing in the ears. In characteristic cases of Ménière's disease these four symptoms supervene suddenly with an apoplectiform attack (accompanied by transient loss of consciousness), which has been shown to be due to hæmorrhage into the labyrinth ; (i.) and (ii.) afterwards become paroxysmal, (iii.) and (iv.) more or less permanent. Knapp has observed that the hearing is impaired at first only for the higher and lower octaves ; and Charcot states that some patients have the vertigo and buzzing only while the deafness is partial, disappearing when this becomes total. This is probably diagnostic of labyrinthine as distinguished from central vertigo. A. Cheatle suggests that the attack may be due to sudden increase in the tension of the perilymph and endolymph.

In regard to *Treatment*, the disease is undoubtedly very intractable, if not incurable, and our efforts should be directed mainly to a reduction of blood-pressure, if this is high, so as to avoid a repetition of hæmorrhage. The symptoms may be relieved by bromides and other sedatives. Charcot recommends 5 grains of quinine thrice daily, and he mentions one case which was cured by this means. Trimelin often relieves the symptoms for a time.   .   '

The patient is attacked with **complete unconsciousness** *suddenly supervening and more or less prolonged.* The case is one of apoplectic or other form of COMA.

§ 564. **Coma** is a condition of loss of consciousness coming on more or less suddenly, in which, in its complete form, all signs of vitality, excepting those of organic life, are suppressed. The patient is deprived of all power of movement and sensation. He cannot hear much less understand an order shouted into his ear to put out his tongue. The raised limbs when let go fall helplessly. The eyelids are closed, and the conjunctival reflexes absent. When the eyes are partly closed the condition is known as *coma vigil.* The respirations are slow and stertorous, owing to the flapping of the palate and the falling back of the tongue on to the posterior wall of the pharynx. The pulse and the breathing are the only signs of life. The temperature is—at any rate, at first—normal or sub-normal. The *typhoid state* (§ 373) is a term applied to the comatose condition which supervenes in certain fevers, and may be recognised by the presence of pyrexia, the tendency of sordes to form about the teeth, and the muttering delirium. *Syncope* is readily distinguished from coma by the blanching of the face, the feeble pulse, and by the unconsciousness being much less in degree and much more transient. The following is a list of the causes of coma :

    I. Head injury.
    II. Cerebral hæmorrhage and acute softening.
    III. Other organic and functional cerebral lesions.
    IV. Alcohol.
    V. Opium.
    VI. Uræmia.

VII. Diabetes.
VIII. Acute yellow atrophy.
IX. Heatstroke.
X. Addison's disease, Raynaud's disease, and other rare conditions.

Coma in children arises under somewhat different conditions (§ 566).

The CLINICAL INVESTIGATION of coma is of great importance, as it is an emergency of the gravest significance. When called to a comatose patient, seen, perhaps, for the first time, the question as to the cause is one of the most difficult we have to solve. The commonest causes of coma are drink, cerebral apoplexy, head injury, opium poisoning, and uræmia, and the prognosis and treatment differ in these several conditions. The mode of procedure should be as follows : (1) *Examine the head* (and other parts) carefully, to see if there be any signs of injury, and if the case be a medico-legal one, make a precise written note on this point. If there be evidence of an injury, the question whether the injury has caused the coma ,or whether it occurred after its onset should be borne in mind. (2) Note the *odour of the breath*. If this be alcoholic, it does not follow that the condition is due to drink, for stimulants are frequently given by the friends to restore the patient. Note also whether the breath has the sweet odour of diabetes. (3) Observe most particularly the state of each *pupil* and the conjunctival reflex. Both pupils are much contracted in opium poisoning and hæmorrhage into the pons. Hæmorrhage into other and commoner situations within the cranium usually cause inequality or dilatation of both pupils. In the other forms of coma they are usually both dilated. (4) Ascertain whether any *paralysis* of the limbs is present, or whether the face is drawn to one side. This may be a little difficult, but usually in apoplexy the paralysed leg and arm of one side of the body are rigid or flaccid as compared with the other side, and by raising the limbs and allowing them to fall it will easily be found whether one side is more rigid or more flaccid than the other. In nearly all cases due to gross cerebral lesion (tumour, abscess, meningitis, etc.), one side is weaker than the other. (5) Count the *pulse*, examine the *arteries*, and auscultate the *heart*. High blood-pressure suggests uræmia, apoplexy, or lead poisoning ; great slowness of the pulse suggests tumours and opium poisoning ; a presystolic murmur suggests cerebral embolism. (6) Count and observe the *respirations*. A stertorous or snoring quality is simply an indication of the profundity of the coma, being due to paralysis of the tongue and palate. The respirations are very slow in opium poisoning. In grave cases of apoplexy and uræmia they assume a Cheyne-Stokes character. (7) Take the *temperature*. It is often very low in uræmia and in opium poisoning, sometimes a little lowered in apoplexy and still more in drink. There is sometimes a tendency for it to run up after an attack of apoplexy, and this is of very serious import. (8) Procure (by catheter if necessary) and examine some of the *urine*. The absence of albumen is against renal disease ; a small amount of albumen does not help one much to distinguish between apoplexy and uræmia. In diabetic urine we find sugar ; in opium poisoning morphia. Atropine and other vegetable alkaloids are also excreted by the urine. (9) Observe the *age*. Coma in childhood is almost confined to post-epileptic coma, meningitis, cerebral tumour, or sinus thrombosis ; about middle age cerebral hæmorrhage is to be suspected. (10) Inquire into the *history*—whether the attack came on suddenly in apparent health or after some previous indisposition. Coma sometimes arises in the course of a disease the history of which is readily revealed by inquiry, such, for instance, as epilepsy. But, on the other hand, the patient's friends may have been quite unaware of the existence of any disease, such, for instance, as diabetes, granular kidney, or cerebral tumour in the frontal region. Convulsions usually precede the coma in hepatic and uræmic poisoning, general paralysis of the insane, and tumours near the motor area of the brain.

The commonest causes of coma in everyday practice are drink, intra-cranial hæmorrhage (with or without injury), opium poisoning, and uræmia (see table on following page).

I. **Head Injuries** may produce either *concussion* (bruising) or *compression* of the brain, the symptoms of which differ more in degree than in kind (table, p. 793). Injury may also be accompanied by conjunctival hæmorrhage, paralysis of the cranial nerves, and inequality of the pupils. Bleeding from the ear and sub-conjunctival ecchymosis do not necessarily (though usually) indicate fracture of the base. Injury to the head may produce compression of the brain, and therefore coma, in four ways : (1) Fracture of the skull, with depression ; (2) hæmorrhage into or upon the brain—in both of which the coma comes on immediately after the injury ; (3) the effusion of inflammatory products, in which case the coma comes on after an interval of a few days ; and (4) abscess, when coma comes on after a week or two at least.

TABLE OF DIAGNOSIS OF COMA DUE TO DRINK, APOPLEXY, OPIUM
POISONING, AND URÆMIA.

	Pupils,(P.) and Conjunctival Reflex (C. R.).	Pulse and Respiration (R.).	Paralysis.	Depth of Coma.	Course.
Drink.	P. equal, normal, or dilated. C. R. present.	Pulse rapid and strong, then weak. R. normal or snoring.	None, but inco-ordination if able to walk or move.	Can be roused.	Progressive recovery in twelve hours.
Apoplexy or Fracture with Compression.	P. usually dilated and unequal. C. R. lost.	Blood-pressure high. R. stertorous ; may be Cheyne-Stokes.	Hemiplegia may be present.	Cannot be roused.	Stationary.
Opium Poisoning.	P. very contracted and equal. C. R. usually present.	Pulse and R. both very slow. R. stertorous.	General weakness.	Can sometimes be roused.	Progressive towards death or recovery in ten to twelve hours.
Uræmia.	P. normal or dilated. C. R. usually present.	Pulse slow and blood-pressure high. R. sighing, and may be Cheyne-Stokes.	None.	Cannot be roused.	oma alternates with convulsions.

§ 565. II. Cerebral Hæmorrhage.—The term apoplexy, or "stroke," is sometimes used to indicate a sudden loss of consciousness due to a vascular lesion—hæmorrhage, embolism, or thrombosis—within the skull. It is, however, more frequently used to denote hæmorrhage from spontaneous rupture of an intracranial artery. The older authors used to state that hæmorrhage from the rupture of a cerebral artery could be distinguished from simple embolism or thrombosis by the occurrence of *loss of consciousness* (sometimes accompanied by convulsions) in the former, and not in the latter. But further experience has shown that this distinction is only a matter of degree ; profound coma may sometimes arise from the embolic blocking of a moderately large artery, or from throm-

Table of Symptoms of Concussion and Compression.

• Note.—There is considerable difficulty in diagnosing whether a case has *slight compression* or a *very grave degree of concussion.*

Concussion of the Brain.	Compression of the Brain.
Symptoms.—As in shock (*q.v.*), *plus* sudden unconsciousness, but patient can be roused in most cases.	Completely unconscious; cannot be roused.
Pupils.—Equal, sluggish reaction to light.	Immobile, often unequal, at first contracted, later on dilated.
Respiration.—Shallow, slow, sometimes sighing.	Slow, stertorous, sometimes irregular.
Motor System.—Muscles relaxed, but no absolute paralysis.	Paralysis; cheeks blown out with expiration; often rigidity on one side of the body.
Bladder.—Frequent micturition.	Retention till overflow with "false" incontinence.

The cerebro-spinal fluid may show alteration in colour, red at first, then golden. The presence of a red or golden hue justifies a diagnosis of hæmorrhage.

bosis; while, on the other hand, slight hæmorrhage, especially if supervening gradually, may be unattended by loss of consciousness. The *extent* and the *suddenness* of the vascular lesion, rather than its nature, determine the occurrence of coma. Cerebral hæmorrhage is most frequent between fifty and seventy, but it may occur even in children.

Symptoms of Cerebral Hæmorrhage.—The attack may be ushered in by a stage of headache or giddiness, lasting a few days and connected doubtless with the associated high blood-pressure; or it may come on suddenly without warning. Convulsions sometimes occur. Sometimes the paralysis comes on with faintness and vertigo only; or it may develop more gradually, followed later by unconsciousness (ingravescent apoplexy). Sometimes it comes on during sleep. If the lesion involves the motor tract it causes paralysis on one side (usually) of the body accompanied by flaccidity or rigidity. The pupils are often unequal, the conjunctival reflex is lost. The temperature, particularly in large hæmorrhages, is usually at first one or two degrees below normal. In the course of twenty-four to forty-eight hours the thermometer usually shows a rise of one or two degrees, at which point it remains for several days. A rapid elevation of temperature within a few hours of the seizure indicates hæmorrhage of the base, and therefore a speedily fatal termination.

Diagnosis of Cerebral Hæmorrhage.—In view of the importance of differentiating this condition in an emergency, a table is given above (p. 792). It should be remembered that cerebral hæmorrhage frequently supervenes in the course of chronic Bright's disease, and therefore uræmia and apoplexy may be concurrent. The diagnostic features of the greatest value are the state of the pupils, particularly their inequality, the loss of the conjunctival reflex, and the augmented blood-pressure. The diagnosis of the various *forms of vascular lesion* is given in tabular form below.

One is fairly safe in excluding cerebral hæmorrhage if the blood-pressure is not high. Though a high blood-pressure is suggestive of cerebral hæmorrhage, it has to be remembered that the arterial degenera-tion which usually accompanies it may give rise to thrombosis as well as hæmorrhage.

As regards the *locality* of the hæmorrhage, the usual position (about 76 per cent.) is the *internal capsule*, from the lenticulo-striate artery (Fig. 159), giving rise to hemiplegia on the side opposite to the lesion. In most of the cases of hæmorrhage into the *ventricles* there is paralysis or rigidity of all four limbs, and the condition is uniformly fatal. Marked contraction of both pupils, or crossed hemiplegia, suggests hæmorrhage into the *pons.* Hurried or Cheyne-Stokes respiration often accompanies hæmorrhage in this position, and the prognosis is grave. *Meningeal hæmorrhage* is suggested by the absence of definite paralysis and the presence of initial and recurring convulsions. *Pachymeningitis hæmorrhagica* gives rise to attacks of coma which differ (in most of the cases I have seen) from the other causes now under consideration in their gradual onset. It occurs most often in lunatics and in the dementia of old age. *Conjugate deviation of the head and eyes* towards the paralysed side is frequent when the hæmorrhage involves the motor tract.

TABLE OF THE DIAGNOSIS OF CEREBRAL HÆMORRHAGE, EMBOLISM, AND THROMBOSIS.

	Cerebral Hæmorrhage.	Embolism.	Thrombosis.
Age.	Middle and advanced age.	Any age, but frequent in early life.	Any age.
Causes.	1. High blood-pressure. 2. Generally after æt. 50.	1. Cardio-valvular lesions, especially mitral stenosis. 2. Thrombus in the peripheral vessels.	1. Syphilitic endarteritis. 2. Cerebral atheroma. 3. Exhausting disease; phthisis; anæmia. 4. Cardiac enfeeblement.
Onset.	Coma usually sudden, sometimes with convulsions.	Sudden onset of paralysis, but usually no loss of consciousness or convulsions.	Often premonitory symptoms, vertigo, convulsions. Coma unusual.

Prognosis.—It is held by some authorities that cerebral hæmorrhage almost always goes on to a fatal termination, and that the cases of supposed cerebral hæmorrhage which recover are really cases of thrombosis. About half the cases of supposed hæmorrhage recover from the attack, but with remaining paralysis. The depth and duration of the coma are fair measures of the extent of the mischief, and therefore of the prognosis. The signs indicating deep coma, and therefore of unfavourable import, are loud stertor, completely insensitive conjunctivæ, flapping cheeks, and increasing cyanosis. Convulsions, or the early appearance of rigidity, or a sudden rise of the temperature, are unfavourable. Coma coming on slowly and progressively increasing (that is, ingravescent apoplexy) is

more unfavourable than that which comes on more suddenly, with less complete coma. A more unfavourable form still is that in which, shortly after the first attack, a second supervenes; from this the patient rarely recovers. As regards *locality*, intraventricular hæmorrhage, basal hæmorrhage, and hæmorrhage into the pons are the most serious.

Etiology.—Cerebral hæmorrhage is more frequent in the male than the female sex, and does not usually occur until after fifty. The rarer cases of " apoplectic seizure " in persons under forty are almost invariably due to embolism or thrombosis. Heredity plays an important part by reason of the tendency to vascular disease which runs in families. Disease of the vessels is an almost necessary precursor to their rupture. High blood-pressure is a most important factor in the causation of apoplexy; it predisposes to arterial disease, and may also determine the hæmorrhage. The causes of high blood-pressure are given in § 72; the commonest cause is chronic Bright's disease. Leukæmia, purpura, and other blood diseases may occasionally cause cerebral or meningeal hæmorrhage.

Cerebral Embolism.—The preceding remarks have reference to cerebral hæmorrhage, but cerebral embolism involving a fairly large artery may give rise to all the symptoms of apoplexy. The age of the patient, and the presence of cardiac disease, especially mitral obstruction and ulcerative endocarditis, aid us in diagnosis (see table, p. 794).

Thrombosis of the Cerebral Arteries.—Thrombosis generally arises from a *gradual* occlusion of the lumen of a vessel by senile arterial disease, or by syphilitic endarteritis in younger subjects. The supervention of symptoms is, however, usually sudden, and is only exceptionally attended by coma (see table on page 794).

Thrombosis of the Cerebral Sinuses may give rise to coma and all the symptoms of apoplexy. It may arise from caries of the skull (syphilitic or tuberculous), extension from a cerebral abscess, *e.g.*, from ear disease, and occasionally from the pressure of an aneurysm, gumma, or other tumour; or in association with meningitis. Sinus thrombosis is favoured by the feeble cerebral circulation characterising cachectic conditions (chronic diarrhœa, enteric fever, and marasmus in children). *Septic thrombosis* and the differential signs of thrombosis of the lateral cavernous and longitudinal sinuses are described under Intracranial Inflammation (§ 586).

The *Prognosis* of cerebral embolism as regards life is usually good, though the paralysis tends to remain. It is most likely to clear up in children. If the causal condition remains a second attack may occur. Cerebral embolism from septic endocarditis is fatal. In thrombosis the prognosis as regards life is good when due to syphilitic endarteritis, less favourable when occurring in the aged, and extremely grave when associated with exhausting disease and anæmia.

The *Treatment of an apoplectic seizure* is not very hopeful. Perfect rest and quiet are very important. The patient should, as a rule, be left in the room where the seizure occurred—a mattress being placed on the floor, if necessary—rather than incur the movement necessary to raise him on to a bed. The head and shoulders should be raised, and the

patient turned gently over to one side to prevent the tongue falling back into the pharynx. The administration of food is, as a rule, undesirable, at least by the mouth, for fear of its passing into the air passages ; and alcohol must be absolutely forbidden. The patient will do no harm for a day or two without nourishment by mouth ; the lips may be moistened. The bladder should be watched, and the catheter carefully passed if necessary. In cases due to *embolism* nothing further can be done except to prevent a recurrence ; in *thrombosis* (other than syphilitic) stimulants are indicated. In *hæmorrhage*—indeed whenever the blood-pressure is high—— a brisk purge is indicated ; two drops of croton oil or 4 to 8 grains (0·25–0·5) of calomel on the tongue may be given, followed, if necessary, by an enema of castor oil or turpentine. The chief indication is to prevent any extension of the hæmorrhage. If the blood-pressure is very high, it is a good practice to bleed to the extent of 10 to 20 ounces. An ice-bag or a cooling lotion to the head is recommended by some. Blisters to the back of the neck, and mustard plasters to the calves or soles of the feet, " to rouse the patient," are worse than useless. As regards the resulting hemiplegia care should be taken to prevent the occurrence of talipes equinus, and permanent flexion of the fingers and wrist, by means of suitable splints and massage.

III. **Other Gross and functional Cerebral Lesions.** -Generalised convulsions are usually attended by partial or complete loss of consciousness (§ 634). Among the **causes of both convulsions** and **coma** are the following :

1. In POST-EPILEPTIC STUPOR the unconsciousness is not so complete as in cerebral hæmorrhage ; except in Jacksonian fits, there is no hemiplegia, and within a few hours the patient wakes. There is usually a history of previous attacks.

2. CEREBRAL TUMOUR and ABSCESS may give rise to attacks of coma, which, in the absence of a history of previous ill-health, are difficult to distinguish from apoplexy. In such gross lesions of the brain there may be (1) optic neuritis ; (2) paralysis of the cranial nerves, and hémiplegia. (3) The coma not infrequently alternates with or is attended by convulsions. (4) There may be a history of headache, giddiness, and vomiting.

3. In one-fifth of the cases of DISSEMINATED SCLEROSIS attacks of coma occur (Charcot), lasting a day or two, and then passing into a state of stupor. In these the face is flushed, the pulse rapid, and temperature elevated to 104° or 105° F. The tremor and other symptoms are always worse as the patient emerges from these attacks.

4. GENERAL PARALYSIS OF THE INSANE at some stage is almost invariably accompanied by fits of various kinds—either of loss of consciousness amounting even to complete coma, or of convulsions with or without loss of consciousness. Attacks attended by loss of consciousness are apt to end fatally.

5. Certain ACUTE and SUBACUTE CEREBRAL LESIONS, such as tuberculous and simple meningitis, cerebro-spinal meningitis, and septic sinus thrombosis, may cause coma. In such conditions it is usually of late onset, and a history and other signs are obtainable (see Intracranial Inflammation, § 582). In cerebro-spinal meningitis lumbar puncture reveals the presence of the diplococcus, and in tuberculous meningitis, of the bacillus. Paralysis is rare, but muscular spasm is common.

IV. **Intoxication,** or Acute Alcoholic Poisoning.—The coma which

supervenes after heavy drinking, or a single large dose of alcohol, may very closely resemble apoplexy and cerebral compression from head injury. In any case of doubt it is wise to admit the patient to the hospital, and to treat him on the supposition of the more serious condition. If house-surgeons would bear this in mind, we should see less of those paragraphs in the newspapers headed "drunk or dying." The smell of alcohol in the breath is fallacious, as friends may have given alcohol to restore the patient. The chief differential features of the coma of intoxication are : (1) The coma is rarely so profound as in apoplexy, and the patient can generally be roused. (2) The absence of inequality of the pupils, hemiplegia, or convulsions (see table, § 564). (3) Procure some of the urine, and add one or two drops of the urine to fifteen drops of a chromic acid solution, made by adding one part by weight of potassium bichromate to 300 parts by weight of strong sulphuric acid : the solution turns a bright emerald green if alcohol be present in quantity (Anstie).

V. In opium poisoning the patient becomes increasingly drowsy, gradually lapsing into (1) coma. (2) The pupils are equal and extremely contracted ; (3) the pulse and respirations are slow ; (4) there is no hemiplegic rigidity or flaccidity of the limbs ; (5) in fatal cases the coma gradually deepens, the face becomes cyanotic, and the pulse and respiration gradually cease together.

For the symptoms of poisoning by other narcotic drugs the reader is referred to manuals on toxicology, but it is well to remember that chloral and chloroform, coal gas, belladonna, cannabis indica, and occasionally hydrocyanic acid, may all act as narcotic poisons.

VI. Uræmia (" serous apoplexy " of the older authors [1]) may lead to coma in the advanced stages of kidney disease. (1) The coma is rarely quite so deep as in apoplexy ; it is more of a stupor or drowsiness. (2) In the majority of cases stupor alternates with convulsions, and in many cases with muttering delirium. (3) There is an absence of hemiplegic rigidity or paralysis. (4) There is albuminuria. It must be remembered, however, that in most cases of sudden coma some degree of albuminuria is present. (5) If a history is obtainable, the earlier symptoms of uræmia will be revealed (§ 296).

VII. Diabetic coma supervenes very suddenly, often in apparent health, and is very profound. Its two characteristic features are (1) the sweet ethereal odour of the breath, and (2) the presence of sugar and ketone bodies in the urine. Unless treated by insulin, it almost invariably results in death, being one of the most frequent modes of termination in diabetes.

VIII. Hepatic diseases which result in widespread destruction of the secreting tissue of the liver—notably the later stages of cirrhosis and acute yellow atrophy—give rise to a condition clinically resembling uræmia. This condition is in most cases

[1] The older authors recognised the clinical resemblance of uræmic coma to the coma of cerebral apoplexy. In chronic Bright's disease the cerebral convolutions waste, and fluid is effused between the brain and the dura ; finding no hæmorrhage this effusion was therefore regarded by them as the cause of coma ; hence the term " serous apoplexy."

differentiated by the jaundice, slight in the concluding stage of cirrhosis, very marked in acute yellow atrophy. The coma under these circumstances has been called cholæmia, under the impression that it is due to the presence of bile in the blood; but bile freed from mucus injected into the blood does not give rise to symptoms. •

IX. **Heat-stroke** (sun-stroke) is classified into (1) a cardiac or syncopal variety, in which the patient suddenly goes off into a dead faint, with symptoms pointing to failure of respiration and circulation; and (2) a "cerebro-spinal" variety in which coma gradually supervenes. The circumstances under which it occurs are the only means of its differentiation. The comatose form is described in § 404.

X. **Certain rare diseases**—*e.g.*, **Addison's disease** and **Raynaud's disease**—are occasionally attended by sudden coma, perhaps with vomiting and hemiplegia. A malignant form of **malaria** (§ 408) is attended with coma which, coming on suddenly, may lack a history; and English-trained medical men newly arrived in India may mistake these cases for apoplexy. *Excessive muscular exertion* has caused coma, probably as the result of accumulation of toxic products (Dr. V. Poore, the *Lancet*, 1894, vol. is, p. 1066).

Fat embolism may cause coma. This form of embolism occurs as a rare complication of fracture, especially compound fracture or fracture of atrophic bones. The condition is attended by dyspnœa, cyanosis or pallor, collapse, cardiac irregularity, and at times by coma and death.

Coma may follow certain forms of *poisoning*, as in botulism, and in the poisoning from infected sausages; in such cases there is a history of gastro-intestinal irritation. Coma may occur in cases of severe anaphylaxis.

The *Prognosis* of coma is always grave, the gravity increasing with its depth and duration. The coma after head injury usually comes under the care of the surgeon. The coma of apoplexy and other vascular lesions has been already dealt with. In post-epileptic coma, if the patient does not recover within a few hours, the status epilepticus is present and the condition is serious. Coma occurring with tumour of the brain or acute lesions is usually fatal. The prognosis of opium poisoning depends upon the amount of the drug administered and the promptitude and efficiency of the treatment. Uræmic coma is not so unfavourable as might be thought; cases recover with proper treatment, but in granular kidney the condition recurs sooner or later. In diabetic coma, which was formerly fatal, the patient now usually recovers with insulin.

Treatment.—Apoplexy has been already dealt with; the treatment of head injury is carried out on similar lines, but here surgical aid may be necessary. Some cases of coma other than apoplexy—fracture of the skull, cerebral tumour, intracranial inflammations, and uræmia—may be relieved, temporarily at any rate, by lumbar puncture (§ 666), and cures have been reported. For alcoholism and all forms of poisoning a prompt emetic should be given; a hypodermic injection of apomorphine is one of the best remedies. Opium poisoning is treated by frequent washing with the stomach-pump. Coffee, atropine, or strychnine is given if the heart or respiration be failing. The patient must be kept awake by walking him about, applying electricity to the limbs, ammonia to the nostrils, and artificial respiration. Diabetic coma calls for skilled and prompt treatment by insulin. For uræmia eliminate the poison in the blood by brisk purgatives, hot packs, venesection, and saline injections.

§ 566. Coma in Children, apart from injury, may be due, in order of frequency, to tuberculous meningitis, post-basal meningitis, suppurative meningitis, post-epileptic stupor, cerebral tumour, syphilitic pachymeningitis, sinus thrombosis, and hæmorrhage; diabetes, abscess, and cysts are rare causes. The history, mode of onset, and associated symptoms aid the diagnosis. Tuberculous meningitis is by far the most frequent cause. Out of 86 cases of coma in childhood, Dr. F. E. Batten found tuberculous meningitis in 50 cases, non-tuberculous meningitis in 17, and cerebral tumour in 16 cases. Cerebral hæmorrhage occurs chiefly in association with the specific fevers, such as small-pox and whooping-cough, also with rickets and scurvy. In marasmic conditions thrombosis of the longitudinal sinus (§ 586) may ensue, together with meningeal hæmorrhage, giving rise to convulsions followed by coma. Thrombosis of the veins of Galen (§ 665) and lateral sinus thrombosis, in association with ear disease, may cause coma.

β. Partial Mental and " One Faculty " Defects

One " faculty " of the mind, such as the memory or attention, may be affected, and there may also be various partial mental defects which do not amount to actual insanity, though they may constitute its earlier phases. These will now be considered—

> Defects of speech (motor and mental).
> Defects of memory (partial and entire).
> Defects of attention (deficiency and excess).
> Defects of other mental faculties.
> Hypnotism.
> Catalepsy and trance.

§ 567. Defects of Speech and other Signs of Thought may be purely motor, or they may be of central or mental origin (ideo-motor or ideo-sensory), but it will be convenient to consider them both here, as they are so frequently associated. All of these defects may be simply and graphically represented in the following scheme :

Defective Communication with others.

Defects in the outgoing processes	.	{ motor defects (paralysis).		
		ideo-motor	. .	{ speech. writing.
Defects in the incoming processes	.	{ ideo-sensory	. .	{ visual. auditory.
		blindness or deafness.		

Motor defects of speech are recognised by an error in articulation. The patient says the words, but pronounces them badly, just as in cases of motor defect of writing the patient can sometimes write, but writes badly. The muscles of speech are those of the lips, tongue, palate, larynx, and respiration, and these, like other muscles, may be the seat of paralysis, tremor, spasm, or inco-ordination.

Clinical Investigation.—In order to investigate a case of motor defect of speech, we must (1) pay attention to each of the different muscles just named. In the course of conversation with the patient we can generally detect any of the motor defects which are mentioned below. If not, we may ask him to repeat the alphabet through,

and we shall readily detect any paresis or other defect of the muscles. (2) Notice whether there be any tremor of the lips or of the tongue when protruded ; any separation of syllables ; syllabic utterance ; or, on the other hand, any slurring or running together of the words. (3) The presence of any hemiplegia, facial paralysis, or other paralysis should be noted. (4) If all these tests be negative, and still the patient is unable to communicate his thoughts properly, turn to Mental Aphasia, § 568.

1. Paralysis of the muscles of the lips, face, and tongue is met with in most cases of *hemiplegia* in the early stage. Here one side of the face is generally obviously affected, especially its lower part, and the tongue, when protruded, deviates to the paralysed side. The speech is thick, and often quite unintelligible. But as the face and tongue recover, the speech returns. This is a pure defect of the muscles of articulation.

2. Partial paralysis of the muscles of articulation is met with in its most typical form in the case of *bulbar paralysis* (glosso-labio-laryngeal palsy) and pseudo-bulbar paralysis. At first, when the tongue is chiefly affected, difficulty occurs with the, linguals, L, N, T, and there is an obvious difficulty in pronouncing words, giving the impression of the tongue being too large for the mouth. Soon after this the labial letters (as P, B, M) also give rise to difficulty ; then the palate becomes involved, the speech becomes nasal, and difficulty occurs with guttural letters—K and G (hard). Finally speech is totally lost (alalia), and the patient can only utter meaningless grunts.

3. A fine tremor of the lips with slurring speech is very characteristic of *general paralysis of the insane.* The syllables are run together as in intoxication— " British Constitution " becomes " Brish Conshon." This defect is also met with in delirium tremens, some cases of meningitis, and occasionally in disseminated sclerosis.

4. A monotonous drawling, indistinct speech is characteristic of *paralysis agitans.*

5. Syllabic or staccato speech characterises some cases of *disseminated sclerosis* ; there is a pause between each syllable, and each syllable is equally accentuated (consti-tu-shon) as in some Latin verse ; hence sometimes called "scanning speech." Occasionally this kind of speech is met with in tabes dorsalis.

6. Stammering is a spasmodic disorder of the muscles of articulation and respiration. The condition resembles a spasmodic tic (§ 630) of the muscles of respiration initiated early in an attempt to speak. Other tics occur in these patients. Most difficulty is experienced with words commencing with explosive or labial letters— B, D, P, T, K, or G. Except in bad cases, the patient can whisper or sing without a defect. In severe cases the spasm tends to spread to other muscles of the face or other parts ; the patient, for example, remaining with his mouth wide open, or his face contorted for an appreciable time before any sound is uttered. The affection appears in childhood, and there is very generally some hereditary defect in the nervous system. *Syllable stumbling* is a variety of stammering in which one or more syllables of a word are repeated. The curability of stammering depends a good deal upon its previous duration of onset. It is worst when it comes on in the adult. The treatment of stammering is a matter for careful education, and there are some good teachers who make the cure of it their speciality. Much can be done by teaching the patient to manage his breath, and always to *take a deep breath before starting to speak.* I have frequently been able to help such patients by instructing them to beat time with their hands, and, without actually singing, to adopt a sing-song method of speech.

7. Lalling or infantile speech is that in which the letters difficult to pronounce— *e.g.*, R, L, C, Sh—are avoided ; British is pronounced Bitty.

8. In idioglossia the child has a speech of his own, which is unintelligible excepting to those accustomed to the child. It is due to an inability to *reproduce the sounds* of words said to him. He has to be taught by a system of lip reading. It may occur in mongolism and cretinism.

9. Rhinolalia operta is the speech met with in cleft palate and paralysis of the soft palate—*e.g.*, diphtheritic—in children. In it the speech has an unmistakable nasal quality. In rhinolalia clausa, on the other hand, which is due to a spasm or

stiffness of the soft palate, the normal nasal quality of the speech is wanting, and this produces the somewhat affected style met with in some hysterical cases.

§ 568. **Aphasia** is, as Trousseau aptly put it, "loss of memory for the signs of thought." In some cases the patient's speech and power of writing are so much deranged that he cannot communicate his ideas to you; in other cases you are unable to communicate with the patient because he cannot ~~recognise words~~ spoken to or written for him.

For speech and writing, the two mechanisms by which we communicate with other people must be intact:

(1) The motor *apparatus* of speech and writing; and

(2) Initiating *centres* in the cerebral cortex which have been gradually educated up to the function of speaking and writing. These centres are appropriately called ideo-motor centres, inasmuch as they subserve the memory of the complicated movements needful for speech or writing.

For the recognition of spoken or written language, the **incoming** processes of communication, two parts also are necessary (Fig. 157):

(1) The sensory *tracts* of vision and hearing; and

(2) Certain receptive *centres* in the cerebral cortex, **ideo-sensory** centres, ~~reared up~~ to the art of recognising ~~and~~ naming words heard and things and words seen (see tables, pp. 802, 803).

In aphasia we are concerned solely with the ideo-motor (or outgoing) and the ideo-sensory (or incoming) centres in the cerebral cortex. Defects of the motor apparatus of speech have been fully considered (§ 567), and defects of the organs of vision and hearing are given elsewhere. In what follows we must assume that both of these are healthy.

(*a*) **Ideo-Motor Aphasia.**—Are the patient's powers of **speech** or **writing** defective ? We will first consider the outgoing forms of aphasia : aphemia (ideo-motor defect of speech) and agraphia (ideo-motor defect of ~~writing~~).

FIG. 157.—APPARATUS OF SPEECH (represented in error in the right cerebral hemisphere).— The auditory and visual ideo-sensory centres and the ideo-motor centres of speech.

The commonest instances of this form of aphasia are associated with right hemiplegia due to lesion of the left cerebral cortex or tissue immediately underlying it, for which reason it is more common in cerebral thrombosis or embolism than in cerebral hæmorrhage, which is rare in the region of the cortex. In certain cases of hemiplegia we find after a time that, although the patient recovers the use of his lips, tongue, and arm, he is still unable to name things properly or to converse, except by writing or by pantomime. This disability may exist in many degrees ; in some cases the patient only occasionally says a wrong word, in others he cannot use a single word correctly. In some of the worst cases the patient makes use of one or two words only (recurring utterances) for all he wishes to say. His *articulation* of those words which he can speak is good ; he has full power in the muscles of the face and the arm, and his hearing and vision are good. But there is a *defect in his mind* which prevents him from recalling the movements of articulation which indicate the word he wants to use. He knows the right word when he sees or hears it ; hold up a pen
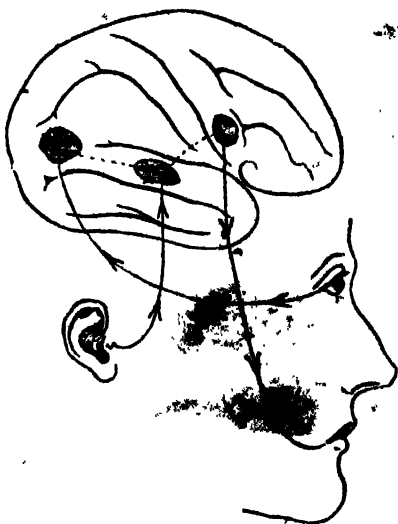
and suggest to him that it is a haddock; he shakes his head and gets angry; write on paper or say " pen," and he nods and smiles. Such a case represents one of the commonest kinds of aphasia; it is a *loss of memory of the co-ordinated movements of speech*, and is known as **aphemia.**

Take another and rarer case, and suppose that, instead of speech, it is only the power of writing which the patient has lost. His motor power is good, his speech correct, and his vision and hearing perfect. But he cannot write a word, although he knows the word he wants to write. In short, he has *loss of memory for the co-ordinated movements of writing*, and the condition is **agraphia.**

Thus we have two kinds of possible defect of a patient's ideo-motor centres in the cortex corresponding to the two principal means by which he communicates his thoughts to the outer world (see table below).

IDEO-MOTOR APHASIA, *i.e.*, defect in the cortical mechanism of the centres of speech and writing.

	Tests.	Nature of Defect.	Position of Lesions in Cortex Cerebri.[1]
Speech loss (aphemia).	Cannot talk correctly or say names of objects.	Loss of the educated recollection of the movements required for spoken words.	Posterior end of third left (in most people frontal convolution (Broca's) and lower end of ascending frontal (Fig. 158).
Writing loss (agraphia).	Cannot write names of objects or convey his thoughts in writing.	Loss of the educated recollection of the movements required for writing.	Posterior end of second left (in most people) frontal convolution.

(b) **Ideo-Sensory Aphasia** (Sensory Aphasia).—Can the patient understand written or **spoken words** ? Let us now consider the afferent forms of aphasia—visual aphasia (word-blindness) and auditory aphasia (word-deafness). They may be unattended by any defect of speech, vision, or hearing for ordinary purposes. But the patient has simply a loss of memory for the signs of other people's thoughts as expressed in written or spoken words.

First, as regards WORD-BLINDNESS (visual aphasia). We have a visual recording-board (Figs. 157 and 158) in the cerebral cortex, upon which our recollection of printed or written words is recorded, so that after seeing them a few times, or being educated to read, we can recognise and name them at once when seen again. If the patient has *lost the faculty of understanding and recognising written or printed words*, the condition is word-blindness (visual aphasia). This form may be met with alone. The vision is good enough for ordinary purposes, but it is a curious circumstance that most of such cases the patient cannot see with the left half of each retina (hemianopsia), because it so happens that lesions in the left occipital lobe which produce word-blindness also cut off the optic radiations which come from the left optic tract.

Secondly, as regards WORD-DEAFNESS (auditory aphasia)—i.e., the non-recognition of spoken words. As in vision, so in audition, we have a recording-board in the cerebral cortex, by which we recognise spoken words. It is by the auditory memory that we learn to recognise such sounds as " mamma " and " papa," and afterwards other sounds in our own and other languages. When a patient's faculty of hearing for ordinary purposes is intact, but *he cannot understand spoken words*, the condition is word-deafness (auditory aphasia). This is the rarest form of aphasia, for the recognition of sounds and spoken language is the first thing we learn, and, as a general

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[1] Note that these centres are ordinarily situated in the left hemisphere, but when the patient is left-handed they are usually situated in the right hemisphere.

IDEO-SENSORY APHASIA, *i.e.*, defect in the cortical receptive centres for word-vision or word-hearing.

Tests.	Nature of Defect.	Position of Lesions in Cortex Cerebri.[1]
Word - blind-ness (visual aphasia).	Can see, but cannot read or recognise *printed* or *written* characters. Usually with left homonymous hemi-anopsia.	Loss of the educated visual memory for written (or printed) signs.	Angular gyrus (visual speech centre); or in the left occipital lobe, so situated as to sever the optic radiations between the angular gyrus and the visual centre.
Word - deaf-ness (audi-tory apha-sia).	Can hear, but cannot understand or recog-nise *spoken* words.	Loss of the educated auditory memory for speech.	Posterior half of the superior t e m p o r o- s p h e n o i d a l (Wer-nicke's) convolution (auditory word centre).

neurological principle, the faculties first to come are less easily damaged than later acquirements. It is practically always associated with word-blindness, and generally also with ideo-motor aphasia.

I have described these four varieties of memory of the signs of thought singly in order to make the subject clear, but they are all interdependent one on another. The complete memory for a pen, for instance, involves a visual recollection of the object and its printed sign, an auditory memory of the sound " pen," and an ideo-motor recollection of the movements used for saying or writing " pen." In point of fact, the four varieties of aphasia which correspond to these forms of memory are nearly always mixed in various proportions, aphemia being the most common, agraphia the next, word-blindness the next, and word-deafness the rarest. This renders their investigation difficult. Par-aphasia was a term used by Kussmaul to indicate a slight degree of mental aphasia with varying admixtures of aphemia, word-blindness, and word-deafness, leading to the misplacing and misapplication of words. Par-agraphia was used by him to indicate slight degrees of mistakes in writing.

Lesions.—Pure cases are, as just mentioned, very rarely met with, but now and then such cases have been observed, and we are thus able to locate the position in the several lesions in the cortex. These are given in the tables, and in Fig. 158. Usually the lesion involves the grey matter of the cortex, occasionally the white strands beneath which unite one speech centre with another. The changes may be structural, as in cases of hæmorrhage, embolism, thrombosis, or tumours; or func-tional, as in connection with migraine, and leave no trace behind them.

Clinical Investigation.—One so seldom meets with aphemia, agraphia, word-blind-ness, or word-deafness alone that the investigation of a case of aphasia is not easy, and it becomes a question as to which of these predominates.

(1) First ascertain whether there is any, or how much, defect in the articulation or motor apparatus of the face, tongue, or arm.

(2) You will find it convenient to proceed next to investigate the patient's power of comprehending your communications to him, in the inverse order of the description I have given above.

(3) Word-deafness, the rarest form of aphasia.—Give the patient some simple spoken order—*e.g.,* " shut your eyes "—but do not accompany your spoken orders by any gesture. If he responds, there is no word-deafness; if he does not respond,

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[1] Note that these centres are ordinarily situated in the left hemisphere, but when the patient is left-handed they are usually situated in the right hemisphere.

he is either deaf (an unlikely thing on both sides), or he is word-deaf; and you must ascertain that he is not deaf by seeing if he turns his head towards a sudden noise or musical sound.

(4) Word-blindness.—Ascertain if his sight is good, and particularly if he appears to be able to see equally well with both halves 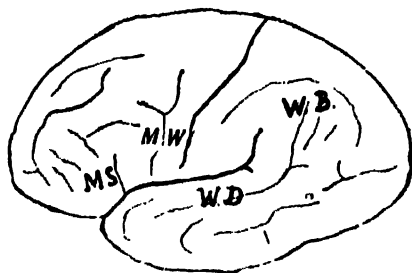of his retina, for word-blindness is commonly accompanied by homonymous hemianopsia. Give him the newspaper (upside down at first), and ask him to identify some of the letters. Then write out some simple instruction and show it to him ; if his sight is good (except for hemianopsia), but he does not respond, there is visual aphasia (word-blindness). In some cases you can place a pen, a penknife, and a watch before him, writing out their names on pieces of paper, and ask him to place them on the objects.

Fig. 158.—M.S., Ideo-motor centre for speech. M.W., ideo-motor centre for writing. W.B., centre damaged in word-blindness. W.D., centre damaged in word-deafness.

(5) Agraphia.—Can he express his thoughts to you correctly in writing ? Ask him to write his name, for instance, or write an account of his illness.

(6) Aphemia.—Can he remember words he wants to say, or does he use them incorrectly in conversation ? Can he name objects correctly ? Hold up a pen and suggest wrong names for it, finally saying or writing " pen," and learn by his gestures if he knows which is the right name.

§ 569. **Apraxia.** The ideo-motor centres for speech and writing are now regarded as forming part of a so-called eupraxic centre which contains the memories for all voluntary co-ordinated movements. There is evidence pointing to such a centre in each frontal lobe (in the intermediate precentral region). By apraxia (or dyspraxia) is meant that a patient is unable to carry out some purposive movement when requested to do so although he may be able to perform the same involuntarily, showing that there is no actual paralysis—e.g., to protrude the tongue or use a pencil. According to Dr. S. A. K. Wilson tumours and vascular lesions in the area of distribution of the anterior cerebral artery of either side and lesions of the corpus callosum mostly produce apraxia, or motor apraxia as it is sometimes designated. Sensory apraxia is due to affection of the ideo-sensory centres, and is for the most part due to imperception (or agnosia), i.e., the failure to appreciate the nature of objects. Both sensory and motor apraxia are frequently to be observed in G.P.I. and in senile and arteriopathic dementia. In right-handed people the left hemisphere is the dominant one, and through the commissural fibres of the corpus collosum influences the right hemisphere, and indeed many movements of the left side of the body are initiated in the left hemisphere impulses passing thence to the right hemisphere. Hence a lesion of the left hemisphere, or of the fibres passing from it to the corpus callosum, may cause apraxia of the left side of the body.

§ 570. Defects of Memory (Amnesia) may be (a) entire—forgetfulness for everything—or (b) partial—forgetfulness for certain facts.

(a) Loss of memory as a whole may be indicated by a forgetfulness for recent facts, such as what day of the week it is ; or for remote events, such as incidents of youth ; or by a forgetfulness of what the patient wished to say or do (intention amnesia), or where he had placed things.

Causes.—(1) *Neurasthenia* and various *debilitating conditions,* such as anæmia, convalescence from severe illnesses, general ill-health, and exhaustion from over-work are commonly attended with some defect of memory. The activity of the memory, like that of the attention (or power of concentration of the thought), is indeed a measure of the activity of the mind, and when the brain is "tired," the memory flags. (2) The memory is also temporarily impaired under the use of certain *drugs—e.g.,* large doses of bromide—but it readily recovers when this drug is stopped. (3) In *advancing years* the memory may become permanently deficient as a normal phenomenon (senile amnesia and senile dementia). The peculiarity of senile amnesia is that it applies chiefly to recent events and to recently acquired knowledge. The patient perhaps can give us full particulars of his early life, and repeat poetry learned in youth, but is unable to mention any event of the same or the previous day. (4) In the mental condition of *chronic alcoholism,* amnesia is a prominent and sometimes incurable condition. For instance, a lady who was under my care for alcoholic neuritis and delirium three years ago, has now recovered both in mind and body, except that she is unable to recall a single fact or incident for five minutes at a time. (5) Defective memory is also met with as a symptom of *dementia* and other forms of chronic insanity. The patient puts things away and forgets where they are ; he also forgets where he is and what he was going to do. (6) *Sudden obliteration of memory* is not unknown. It may occur after a severe illness, and a portion or page of the recollection becomes blotted out, the patient picking up the thread of his life where it left off ten or twenty years before. The case is mentioned by Sir William Gowers of a clergyman aged sixty who obstinately believed he was forty, and picked up the thread of his life at that age. Sometimes such sudden obliteration occurs after epilepsy or without known cause, as in cases reported in the papers from time to time of persons who have forgotten their name and all particulars about themselves.

(7) *Dual personality* (Synonyms : "dual consciousness," "alternating conscious-ness") is another mental condition, in which loss of memory is a prominent feature. It is a rare and interesting condition, in which a patient lives alternately two different lives, or rather, has two alternating mental states, which may for purposes of descrip-tion be called State A and State B. The essential peculiarity of the condition is that the patient when in State B has no recollection of his thoughts and acts while in State A, and *vice versâ.* In each state he picks up the thread of his life when he was last in that state. Moreover, while in State B he may behave in a totally different manner to that in which he behaved during State A. He is subject, in other words, to alternating states of consciousness and character, in which the whole mental attitude and mental record is changed. As a temporary condition dual consciousness may occur after epilepsy (masked epilepsy, or minor epilepsy chiefly), or after hysterical attacks. As a more permanently alternating condition it is found in some without this association.

(b) Among the partial losses of memory—
Mental aphasia may be mentioned. It is, as we have just seen (§ 568), a loss of memory for the signs of thought.

Verbal amnesia is really a slight degree of ideo-motor aphasia manifested by an inability to recollect the words or names when the patient wishes to speak. It occurs with a tired brain or after some emotional shock.

Visual and Auditory Amnesia.—Many interesting observations of late years go to show that the memory is compounded of different constituents. Apart from the association of ideas, there are two distinct ways by which different individuals recall

an idea. (i.) Some recall an idea by a visual impression, such as the form or colour of the object it represents, or by the image of the printed word representing it which they have seen or read. This is called a *visual memory*, and people who chiefly use this form, remember those ideas best which were originally conveyed to them by pictures or objects seen, or by reading a descriptive passage to themselves. (ii.) Others can best recall an idea which is conveyed to them through their ears—*e.g.*, by sounds or by words read aloud to them—and these have what is called an *auditive memory*. Though everybody possesses both these forms of memory, they unconsciously make use more of one than the other. The visual memory of most people is better than their auditive memory, and they recall objects and pictures seen much more readily than sounds heard ; hence the great value of kindergarten and demonstrative methods of education. Charcot narrates a remarkable case of a highly educated man, a banker, who, having an unusually good visual memory, suddenly, after a severe emotional shock, lost it completely. He was conscious of a great blank in his mind, and was unable to picture to himself any of the forms with which he had formerly beeñ familiar, such as the shape of a building, a column of figures, the colour of his wife's hair, etc. By degrees, however, he learned to substitute his auditive memory, which hitherto had lain dormant, and so he gradually became able to carry on his business again.[1]

γ. *Acute Perversions of the Mind* (*Delirium and Mania*)

§ 571. Acute mental exaltation or excitement occurs clinically in two forms—delirium and mania—which differ, however, less in their clinical features than in the circumstances under which they occur. I. *Delirium* is the term applied to mental excitement which is clearly traceable to some bodily disorder of which it is a symptom or complication. II. *Mania* is the term applied to mental excitement when no such source can be traced, the mental condition being the only—or, at any rate, the principal — symptom. III. *Active* or *acute* melancholia is occasionally met with ; it only differs from chronic melancholia (§ 574) in the misery and depression of the patient being of a more aggressive character. Mania is practically always acute ; melancholia nearly always chronic. Both are liable to relapse. The one frequently follows on the other, and by many authorities they are considered as phases of one disorder—Manic-Depressive insanity (Kraepelin). When hallucinations and illusions prevail together with symptoms of exhaustion, the condition is frequently called Confusional insanity.

Clinical Investigation.—The first and most important point in any given case of delirium or mental excitement to which you may be called for the first time is to ascertain the temperature, for the most useful clinical division of the causes of delirium is into *Febrile* and *Non-febrile*. Secondly, it is important to make a thorough and complete investigation of all the organs of the body, to ascertain whether there be any local inflammatory disorder, such as pneumonia, with which delirium may be connected, either directly or indirectly. I remember once overlooking a case of latent pneumonia, and consigning it to the lunatic ward of the workhouse. The urine also should be carefully examined for albumen, sugar, or other abnormality. Thirdly, an inquiry should be made into the history of the malady and of the patient, especially as regards alcohol. In reference to the etiology of delirium, three important *predisposing causes* have to be borne in mind. First, there is a marked predisposition in some nervous people to develop delirium in presence of a slighter cause than would

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[1] " Leçons Cliniques sur les Maladies du Système Nerveux," tome iii., Leçon 13.

be operative in others. Secondly, there is a marked hereditary tendency towards the same vulnerability ; and thirdly, excessive drinking predisposes to the occurrence of delirium after an injury, operation, and many diseases which are not usually so attended.

I. The causes of delirium have been considered (§ 372), and need only be here enumerated.

Febrile.	*Non-febrile.*
Diseases of the brain especially meningitis.	Delirium tremens.
Acute visceral inflammations.	Chronic renal disease.
Acute specific fevers.	Post-febrile delirium.
Delirium tremens (rare cases).	Reflex delirium.
	Deliriant drugs.

II. **Acute Mania** may supervene suddenly-- (1) during convalescence from exhausting diseases (as previously mentioned) ; (2) in the course of other diseases of the nervous system--e.g., G.P.I. ; (3) in the course of some other form of insanity. Its onset is usually rapid, tongue-tremor being often met with in the early stage. The stage of excitement is soon reached—loquaciousness, sleeplessness, continual restlessness, incoherence, in which delusions and ideas succeed each other with great rapidity, sometimes relating to moral and religious, at other times to intellectual topics. After lasting some weeks or months, recovery (sometimes quite suddenly) ensues ; sometimes it is followed by moral or mental obliquity or dementia ; rarely it passes into chronic mania. The temperature is normal throughout. In many cases there is a tendency to relapse and sometimes an alternation with melancholia.

Acute Delirious Mania (Bell's Mania) is an acute maniacal condition coming on suddenly in a person in apparent health, attended by pyrexia, usually running a rapidly fatal course, no lesions being found after death. It is happily a somewhat, rare disease. The symptoms come on abruptly, and quickly amount to frenzy, accompanied by outbreaks of great violence and refusal of food. The temperature ranges irregularly from 100° to 104° F., and in the course of one to three weeks the disease terminates in great bodily prostration, and usually in death. Acute delirious mania differs from acute mania in the elevation of temperature, the rapid wasting, and its more rapid and fatal termination. It resembles some cases of enteric fever very closely, acute pneumonia and acute meningitis, but their proper symptoms are absent.

The *Treatment* of mania consists mainly of rest in bed and in the administration of food (with stimulants if the pulse so indicates). Narcotics and depressants may be tried, and Sir G. Savage mentions a case which apparently got well under frequent small doses of opium. For acute delirious mania the wet pack or the graduated bath (§ 422) may be given. In most cases tube feeding is requisite and if insomnia is persistent the administration of chloroform may be indicated.

III. **Acute dementia** (" stupor ") is a rare condition of sudden ablation of the mental faculties.

δ. Chronic Perversions of the Mind (Insanity)

§ 572. This is a more complex group than any of the preceding, and it forms the collection of morbid conditions of the mind, known as insanity. The subject may be briefly dealt with under—

Special types of insanity, such as G.P.I. ; delusional insanity and paranoia ; dementia præcox ; epileptic insanity ; hysterical insanity, including

catalepsy and trance; moral insanity; obsessional and impulsive insanity; alcoholic, syphilitic, and puerperal insanity .. § 576 and § 577
The mental defects special to children and adolescence .. § 580 and § 581

CLINICAL INVESTIGATION.—I have already, in § 546, referred to the importance of tact and general knowledge in investigating physical disorders, and I may here mention two other points : (1) Get your data in chronological sequence as far as possible ; and (2) never be in a hurry. Any careless, inept, insistent, or rapid questioning will only confuse and silence your patient and defeat your object. Gain his confidence ; let *him talk to you* first of his favourite hobby, then of his thoughts and feelings, and finally of his delusions and hallucinations. The main points to investigate in mental cases are sleep (and dreams); writing; memory; decision and will; reasoning power; moral and ethical standards; delusions : hallucinations; and, lastly, whether the patient regulates his conduct according to these—*i.e.*, whether he (or she) is a potential danger to himself or others. Before committing yourself to an opinion never omit to ascertain from the relatives how far the patient's present differs from his previous character and conduct, for conduct that is mad in one person is normal in another.

The three terms, *delusion, illusion,* and *hallucination,* strictly speaking, have different meanings, but they are used somewhat laxly and indifferently. A hallucination is a false sense perception without any external stimulus—*e.g.*, the hearing of voices and the seeing of snakes. *Illusions* are distortions of sensory perceptions, as in alcoholic delirium, when a lady's muff is thought to be a cat. A *delusion* is a false idea or judgment which cannot be accepted by people of the same class, education, race, and period of life as the person who expresses it. It is predisposed to by a state of depression or of elation, and may be excited by hallucinations. Hallucinations are met with particularly in exhaustion, mania, delirium, mania *e potu* and paranoid conditions ; it is surprising what minute details can be given to us about these creations of the mind. Hallucinations of sight (rats and snakes) are much less common than those of hearing (hearing voices). Hallucinations of taste and smell are present not infrequently in association with delusions of being poisoned. Hallucinations of common sensation are both frequent and various, and are especially frequent at the climacteric and in hysterical subjects who become insane. Amongst the varieties of sensation hallucinations of the male or female sexual organs are not infrequent, and in this way false accusations may be made against those (especially doctors and nurses) with whom the patient comes in contact.

In regard to all delusions, illusions, or hallucinations, it is important to estimate as far as possible how far such perversions of the mind influence, or are likely to *influence, the acts or conduct of the individual.*

After noting the age of the patient, the first question to investigate in the history is whether the attack came on with excitement or with depression. If with excitement, the case may be one of mania or general paralysis of the insane or delirium (§ 571). If the case came on with depression, it may be melancholia or G.P.I. If the patient is an adult, start at § 573 ; if an adolescent, at § 580 ; if a child under ten, start at § 581.

§ 573. I. Chronic **Mania** is simply a prolonged form of acute mania, as previously mentioned (§ 571), lasting with less excitement for years, instead of for weeks or months. Recurrent mania is that which recurs, sometimes at the menstrual epoch. " Folie circulaire " is an alternation of mania and melancholia, with lucid intervals. Monomania was used by Esquirol to indicate a form of delusional insanity (§ 577) dominated by one fixed idea ; it is not really mania.

§ 574. II. Chronic **Melancholia** is a morbid condition of miserable self-consciousness and self-abnegation without hope. Melancholia occurs under three circumstances : (1) It may be part of some other mental disorder, such as G.P.I., or a stage (first or third) of mania ; (2) it may constitute the whole of the mental disease without previous ill-health ; or (3) it may supervene on neurasthenia or some bodily ailment.

The onset is usually insidious, and commences with extreme self-consciousness, combined with sadness, as indicated by depression, without cause, and when the patient is remonstrated with he is irritable. There are morbid dreads of impending calamity which cannot be named, sleepless nights, and a suicidal tendency. Among the physical signs commonly noted in such cases are feeble circulation, as evidenced by cold feet and chilblains, and constipation. Melancholia differs from hypochondriasis in the "hopelessness" of the former and apathy to surrounding conditions.

Four *varieties of melancholia* are described—active, passive, and simple melancholia, and melancholic stupor. (1) and (2) *Active* and *passive* melancholia depend upon the degree to which patients give expression to their grief. In the former they are always imparting their trouble to some one; it is in reality an acute melancholia. In the latter they sit for hours together in a dejected state, and it is sometimes difficult to elicit their leading delusion. (3) *Simple* melancholia may be described as melancholia without definite delusions. It consists simply of misery, sleeplessness, self-blame, and inability to continue at work. This form is common in the overworked or much-worried, and in women at the climacteric. Suicide is not uncommon in these cases, and precautions, which are sometimes neglected on account of the simplicity of the affection, should not be omitted. Otherwise the prognosis is favourable. (4) In melancholia with stupor (melancholic stupor) the patients remain speechless and motionless, with an aspect of abject misery. Their limbs may be flaccid or in cataleptic rigidity. They are abstracted and oblivious to all external stimuli. They resist external interference, but are not usually violent. The condition may come on suddenly or gradually, and last for months or years. Some cases end fatally in a short time. Some have recurrent periods of exaltation. Some terminate in a condition of permanent weak-mindedness, and a few recover. It is equally common in both sexes, but is more frequent in the young than in the old. Sometimes it follows a severe and exhausting illness, and sometimes it follows acute mania.

Course and Prognosis.—The melancholic process is longer than the maniacal one. The duration varies considerably, but lasts an average of some three to twelve months. Relapses are not infrequent. The slower the advent of the disease, the slower is the recovery. Recovery is common enough in the young, the prognosis being worse as age advances. Suicide is frequent in all forms, but death from the disease is rare. Heredity is an important factor, and the nutrition of the body at the time is another. The melancholia of pregnancy is favourable, but the melancholia of lactation is always grave. It is interesting to remember that cases of melancholia may recover even after a very long time (nine, ten, and thirteen years have been recorded). There is a distinct *suicidal tendency* in all cases of melancholia, but those are specially liable who have delusions of impotence, of being followed or persecuted, of hearing voices, of being the cause of injury to their relatives, or who are suffering from great physical weakness or bodily disease. The tendency to suicide appears to increase with age, and suicidal cases generally have an insane heredity, and often a suicidal heredity also. Patients generally have a special predilection for some particular mode of death—one to poisoning, another to drowning, another to hanging, and another to blowing his brains out, and they will often avoid other means which may happen to present themselves.

In regard to *Causation*, melancholia depends in most cases more upon physical and external than upon mental and moral causes. Some of the commonest causes have been referred to under the variety Simple Melancholia. No doubt want of society, solitary habits combined with a sedentary life, in which the person is debarred from genial companionship, are the prominent causes of this condition. A general depression of the vital powers—*e.g.*, from bodily disease, fevers, heart disease, etc.—is an important factor. Any age may be affected, but it mostly arises at or after middle life.

Treatment.—In the simpler cases, such as those referred to under Simple Melancholia, a few weeks' rest under supervision, with a pleasant companion and complete absence of the conditions under which the disease arose, followed by a few months'

easy travel, will generally set the patient right. If the interest can be aroused, and the attention attracted for a sufficient time, much can be done to relieve the condition and even remove the delusions. Feeding is necessary, and in case of refusal it may be done by means of (a) a spoon, pouring the fluid into the cheek beside the teeth, or (b) by the nasal or stomach tube. The quantity thus administered should be equal to 5 pints of milk, 2 pints of strong beef-tea, 6 eggs, and 3 to 6 ounces of brandy per diem. Insomnia may require the administration of a hypnotic at night time. Suicide must be prevented by removal to an asylum or careful watching at home.

§ 575. III. **Dementia** is deficiency of all the mental faculties, coming on in adult life. It comes on as a *primary* condition in (a) chronic alcoholism, and (b) advanced life (senile dementia). The first, as we have seen, shows itself especially by a loss of memory. The second also has the same peculiarity, with the additional feature that the memory is lost for recent events only. Dementia comes on as a *secondary* condition in (a) general paralysis of the insane, and as the concluding stage in many other forms of mental disease, notably dementia præcox; and (b) after vascular and other gross intracranial lesions. Even after a small lesion of the brain the mental capacity for business is hardly ever as good as before its occurrence, and the patient often becomes childish, peevish, forgetful, emotional, and by degrees in severe cases, completely demented.

The **Special Forms of Insanity** are general paralysis of the insane; delusional insanity and paranoia; dementia præcox; epileptic insanity; hysterical insanity; catalepsy and trance; moral insanity; obsessional and impulsive insanity; alcoholic, syphilitic, and puerperal insanity.

§ 576. IV. **General Paralysis of the Insane** (G.P.I.; Paralytic Dementia) is a progressive generalised muscular weakness and tremor, accompanied by mental symptoms, often of a grandiose character, occurring almost entirely in young men or men in the prime of life; due to atrophy and a scattered sclerosis of the cortex cerebri. Until quite recently, the disease was considered to be due to a parasyphilitic process. The spirochæta pallida has recently been detected in the cortex of the brain and in the subcortical tissues, at some distance from the blood-vessels. What used to be called parasyphilis is now known to be syphilis of the nerve elements themselves, whereas cerebro-spinal syphilis is syphilis of the interstitial structures, *i.e.*, blood-vessels, membranes and neuroglia.

Symptoms.—Paralysis of the limbs may sometimes exist for many years without mental symptoms (*vide infra*). In my own experience, mental have generally preceded the physical symptoms, but this order varies. The characteristic symptoms are mental alteration, general weakness, tremor, and alterations in the pupils and the speech. They are divided for convenience into three stages, each of which lasts about one year. In the first or *premonitory stage* (the stage of irritability and muscular tremor) irritability, restlessness, perversion of the moral sense, and loss of the faculty of attention are among the most usual features, though a variety of other mental aberrations are met with. A man of even temper, who has been a fond husband and father, becomes irritable over trifles, gives way to coarse and blasphemous language, exhibits sexual aberrations, or commits thefts. He becomes egotistical, showing the delusions of grandeur so characteristic of the malady, and squanders his money. He

may believe himself to be very strong, very wealthy, or very high born. Sometimes, on the other hand, great depression is the characteristic of this stage ; and thus, even in this period, we find two groups, one with a tendency to expansion and restlessness, the other with a tendency to despondency and loss of energy. Accompanying, preceding, or following the mental symptoms are various *physical changes*, amongst the commonest of which are (i.) tremor (fine, small, and rhythmical) of the hands (giving rise to characteristic handwriting), and of the lips and tongue (giving rise to a very characteristic speech—viz., a slurring of the words as in intoxication). (ii.) The pupils in this stage are usually small, very contracted, and immobile to light (" pin-point pupils "); very often unequal. (iii.) Headache, neuralgia, and various subjective sensations are sometimes complained of. (iv.) Weakness of the limbs, always generalised, and sometimes combined with some inco-ordination. As a rule, the knee-jerks are increased in G.P.I. Sometimes the symptoms of lateral or posterior sclerosis are present. Anæsthesia and other alterations of sensation and of the special senses are frequent. The *second stage* (stage of fits) is characterised by (i.) mental enfeeblement, which replaces the exaltation of the first stage ; (ii.) increasing muscular weakness, difficulty in walking any distance, and especially in the act of turning, sometimes combined with giddiness ; (iii.) fits are almost invariably present at some time during this stage ; they vary in character, but are usually syncopal or epileptiform, with or without the loss of consciousness. Sometimes they consist of attacks of numbness of the limbs, or aphasia, or coma. The *third stage* is the stage of progressive mental extinction. The speech becomes inarticulate, the paralysis extreme, and may be accompanied by contracture, so that the patient cannot feel himself. His mind undergoes progressive extinction, and there is loss of all its faculties. The urine and fæces are passed involuntarily.

Many different varieties have been described, but these only exist in the earlier stages of the disease ; they all tend to one common form of progressive mental enfeeblement. (1) The expansive variety is the commonest, and forms the basis of the above description. (2) The melancholic variety is characterised by great depression and passes into stupor, or has maniacal symptoms before dementia supervenes. (3) In the paralytic variety, paralysis and tremor predominate, with few or no mental symptoms, excepting occasional outbursts of emotion and some change in character. Sometimes the paralysis predominates in the legs (ascending variety) ; the legs may be rigid (spastic form) ; and there is an ataxic form closely resembling tabes dorsalis at the outset. (4) A congestive variety has been described, chiefly characterised by fits of various kinds. (5) A juvenile variety, between the ages of fifteen and twenty-five, due to congenital syphilis.

Course and Prognosis.—The duration varies widely from a few months to three or more years, and the proportion occupied by these various stages differs greatly. One thing is very characteristic of the disease—

remarkable intermissions of comparative or complete return to health. But these cases always break down on attempting to resume their former state of life. The expansive form above described usually runs its course in about three years. Where depression and melancholia are marked features, the prognosis is worse, and the disease is more rapidly fatal. The spinal paralytic or ascending form occupies a much longer period, and may extend to six or eight years. When the malady is once established it invariably progresses towards a fatal termination.

On account of its great variety, G.P.I. has to be *diagnosed* from many - different complaints : (*a*) From other forms of *mental disorder*, especially alcoholic insanity, chiefly by the tremor, speech, the pupillary changes, and the progressive lethal paralysis ; (*b*) from other diseases giving rise to *generalised paralysis* (see § 609) ; (*c*) maladies attended by tremors and other neuro-muscular symptoms, such as *disseminated sclerosis* and *paralysis agitans*. Chronic *alcoholism* and *peripheral neuritis* are difficult to differentiate sometimes ; they are recognised by a history or evidence of alcoholic dyspepsia and by absent knee-jerks. *Bulbar paralysis* is recognised chiefly by its symptoms being confined to the mouth, tongue, and throat, the mind being usually normal. Lumbar puncture (pp. 952 and 956) greatly aids the diagnosis of G.P.I. ; there is lymphocytosis, increase of globulin, and in 97 per cent. of cases a positive Wassermann reaction, whilst the discoloration of the test tubes in the Colloidal Gold reaction is characteristic. Gross *cerebral syphilis* may be difficult to differentiate clinically ; the cerebro-spinal fluid, however, is not positive to Wassermann's test. The diagnosis from *tabes dorsalis* is not usually difficult, but these two diseases are very apt to occur in a mixed form.

Causation.—(1) Adult males, in the very prime of their strength and manhood—that is, between thirty and forty—are the favourite subjects of the disease, but it may occur at any age. There are congenital cases. It is generally said to be four times as common in men, but I believe it to be commoner. It is more frequent in the lower classes of life. A neurotic heredity is said to be in operation in as much as 30 per cent. of the cases. The disease is in its essence a syphilitic process attacking the nerve elements, resulting in a secondary toxaemia in which other organisms may take part. Alcoholic, sexual, and other excesses, anxiety, and mental fatigue are accessory causes.

Treatment.—Something may be done in the way of *prevention* when there is a history of syphilitic infection by the avoidance of overstrain and of alcohol. Patients with a hereditary taint and premonitory symptoms such as the above should certainly avoid matrimony. The most important *remedial* measure consists of the removal of the patient from the conditions under which the disease has arisen, and especially avoidance of business and all causes of anxiety or mental strain. He should live a regular life, with outdoor exercise and amusements which take him out of himself. Iodides and nerve tonics (especially cod-liver oil) sometimes do good in the earlier stages. When excitement is present, the head

should be kept cool (possibly with an ice-bag) and the feet warm, and the bowels should be freely opened. Physostigma or hyoscyamin may be given ; and if much excitement is still present, 30 minims tinct. digitalis every four hours, or a warm bath followed by an ice-bag. The cold pack is also useful, and bromide and sulphonal. In the melancholic varieties, arsenic, iron, and quinine are the only remedies that have been found useful. Galvanism to the central nervous system has not, so far, been attended with much success. Ordinary methods of administration of salvarsan and antisyphilitic remedies give but little result when the disease is established, because the spirochæta lie so far from the blood-stream. Satisfactory results are obtained by withdrawing blood after a dose of salvarsan, then injecting the serum into the spinal canal. Experience will decide in time the best method of intrathecal medication. The best results have followed the introduction of the remedy directly into the lateral ventricle of the brain,[1] but latterly even this treatment has not come up to the expectations of its advocates. Urotropine (gr. x. bis die) has a sterilising effect on the C.S.F. and often does good ; another therapeutic measure which has been used with some success is the intra-muscular injection of nucleinate of sodium (2 grammes to 100 c.cm.). The latest treatment is subcutaneous injections of 5 c.c. of blood (which has been citrated) from an active case of tertian malaria ; this dose may be repeated if necessary, and then quinine is administered.

§ 577. **Other Special Types** of insanity are named according to their clinical features, such as delusional, hysterical, and moral insanity ; or according to their etiology, such as alcoholic, syphilitic, and puerperal insanity.

V. **Delusional Insanity** is a chronic form of insanity, in which the leading or sole mental alteration consists of a fixed delusion or hallucination, which modifies the conduct of the individual (compare Clinical Investigation, § 572). A delusion, illusion, or hallucination may arise under three conditions : (i.) It may arise in a person who is otherwise perfectly sane ; (ii.) it may be associated with other evidences of insanity, or be a sequela of a past attack ; or (iii.) it may, when no other symptom is present, constitute in itself delusional insanity—when, that is to say, it controls the conduct of the individual. Delusions, especially on religious subjects, are not at all uncommon in the so-called sane. But when these delusions modify the acts or conduct of the individual and lead him to act in an unusual manner, the condition becomes one of insanity.

Paranoia is the modern term used for a variety of insanity in which the patient's whole mental life is dominated by a delusion—usually a fixed one of persecution. Disorder of judgment is the characteristic feature, and in consequence the patient interprets every incident which he observes or takes part in as fresh proof of a plot against him. There are two classes of paranoics. In the first, which is of a milder character and rarely needs asylum care, the patient's own personality does not take any part in the delusion, but he is possessed by some wild theory which he preaches in and out of season ; in the second class, which is a grave form of insanity, the patient's own personality is all-important, and delusions of persecution are common. This delusion is liable to lead the patient to assassination of some prominent person or even to attempt suicide in order to call attention to his case. Megalomania is apt to develop as the disease progresses. Hypochondriasis, in which the patient's attention is focussed on his health or lack of it, is sometimes a sub-variety of paranoia, but does

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[1] Dr. Harry Campbell, the *Lancet*, May, 1914.

not lead to any disorder of conduct likely to cause harm to the community. *Folie à deux* is a condition in which one patient, usually a paranoic, persuades another with whom he or she is very intimate of the reality of the supposed plot against their lives or characters. The second patient, sometimes called the passive element, is then insane, but is more likely to recover. In true paranoia there is no recovery, although occasionally a remission may occur. When the paranoic disorder is dominated by active hallucinations the condition is called **Paraphrenia**, and mostly arises in past mid life.

VI. **Dementia Præcox.**—This is a process of mental dissolution, appearing in persons predisposed to this form of insanity, usually between the ages of fifteen and thirty years. It comprises about one-eighth of all the admissions to asylums (Stoddart). A history of some form of insanity, often of dementia præcox, in the family is the rule, and the patient often shows one or more stigmata of degeneration— *e.g.*, deformities of the ears. The general health is poor ; appetite is lost, and constipation and amenorrhœa are usual. The forehead is markedly wrinkled, far more than in melancholia ; occipital headache is often complained of, and the tendon reflexes are apt to be exaggerated. These pass off as the case becomes chronic and gains flesh and bodily health under institutional treatment. The usual mental state is one of complete indifference to the surroundings in the hebephronic variety (**Hebephrenia**) ; hallucinations and delusions are prominent in the paranoidal variety (**Dementia Paranoides**). There is often present one of the following symptoms : Flexibilitas cerea, a condition on which the limbs remain for a long time in any position in which they are placed ; Echopraxia and Echolalia, in which the actions or words of bystanders are imitated although questions are not replied to ; Katatonia, in which the patient stands rigidly all day in one position unless he is disturbed ; or Negativism, a condition in which the patient does the exact opposite of anything that is required. There are many other disorders of conduct almost pathognomonic of Dementia præcox, but for these the reader must consult one of the larger textbooks on Insanity. The disease is incurable except in very rare instances.

VII. **Epileptic Insanity.**—About 10 per cent. of epileptics become so far unmanageable as to be regarded as insane. The mental aberration may be (1) pre-paroxysmal, (2) post-paroxysmal, (3) associated with petit mal only or as an epileptic equivalent, or (4) a general mental deterioration.

VIII. **Hysterical Insanity.**—The mental perversions to which hysterical subjects are occasionally liable are (1) emotional states ; (2) hystero-epilepsy with insanity ; (3) ecstasy ; (4) catalepsy ; and (5) trance. The first is an emotional condition often of a religious kind—a religious veneration for the curate, for instance. The patients are rarely or never suicidal or melancholic, though they may be passionate, mendacious, mischievous, crafty, noisy (screaming and singing hymns), and given to various kinds of movement, such as hammering and dancing, or to the striking of attitudes (as in ecstasy). Perversion of the tastes and the appetite and a general capriciousness are very characteristic.

Cases of the class under consideration are relatively frequent, and in general terms the *Prognosis* of such cases is more favourable than many other forms of insanity, especially if no hereditary mental taint exists, and due skill and judgment are employed in their treatment. In the *Treatment* of these hysterical mental affections we should, as far as possible, avoid putting them to bed, for complete want of will—that is, lack of energy and initiative—is a characteristic feature, and they will soon become bedridden. Change of environment, interests in life, and judicious companions are the central points. It is seldom necessary to send them to an asylum. Artificial feeding may be required, but should be discontinued as soon as possible. Savage suggests adding salt to create an artificial thirst, and then placing fluid noirishment in the way of the patient when, as sometimes happens, she is too lazy to feed herself. Laziness leads to dirty habits and the passing of fæces in the bed. We should look out for tubercle in these cases. Organic disease of the brain has sometimes been found.

Catalepsy (καταληψις, a seisure or attack) may be defined as a state of stupor

in which the patient is deprived of sensation and voluntary motion, in which the limbs remain in any position in which they are put (flexibilitas cerea). The patient may appear, but it does not follow that she is, unconscious of her surroundings. The eyes may be open, but she appears totally oblivious to all the outside world, and she may lie for hours, or days, perhaps, passing her motions under her. These patients are nearly always of the female sex, and are invariably the subjects of some other hysterical manifestations, to which category, indeed, the condition belongs. Sometimes these cataleptic attacks are ushered in by hysterical convulsions or a hysterical faint. They are usually determined by a fright or some emotional storm.

Trance is a condition of stupor allied to catalepsy, in which the limbs are either rigid, or, more usually, flaccid, and lacking the feature of remaining in any one position in which they are placed. The patient may remain for weeks or months in what seems to be a faint, taking no notice, eating no food, making no movement, and scarcely breathing ; though here again she may not be as unconscious as she seems. The pulse is hardly preceptible at the wrist, and unless the patient is forcibly fed she may ultimately die ; but it is surprising the length of time she may live with hardly any nourishment.

IX. **Moral Insanity** is recognised by some as a special form of insanity, in which the mental disorder consists principally, and sometimes solely, of a marked deflection from the normal standard of morality. The intellect and the will may be normal, and the emotions under control. In the adult, moral and ethical perversions occur (1) very frequently as an *early phase* of G.P.I., and many other forms of insanity, for, as Esquirol remarked, moral alienation is but the "first step to madness." (2) It is also met with in the adult *after recovery* from an attack of mania or other mental disorder. The patient, especially if young, is frequently left with a sort of moral scar, and the lower or animal side becomes prominent. (3) Occasionally one finds in adults a moral defect as a *substantive condition* without previous insanity and without any other mental defect. But often the childhood of such patients presented some similar defect in a less degree, for the condition is more frequently hereditary and congenital. *Kleptomania* (in which the patient is afflicted with an irresistible impulse to thieve) belongs to this class. Other patients have *irresistible erotic or amorous* tendencies, and others have a tendency to unnecessary exaggeration and lying. *Dipsomania* is a paroxysmal, irresistible craving for drink. (4) *Moral imbecility* as a substantive disorder is chiefly met with *in children*, particularly those of alcoholic, insane, or epileptic parents. In early childhood they may be perverse, mischievous, cruel, untruthful, or thieving. They are often precocious, and they may even be intellectually gifted. Nevertheless, such children generally need special care under the Mental Deficiency Act, or else they will find their way into prison. It seems probable that a certain proportion of the criminal convictions among the children of the lower orders are subjects of this malady. The condition, as a rule, is incurable, unless by educational and disciplinary measures (*vide* §§ 580, 581).

X. **Obsessional and Impulsive Insanity** (pyschasthenia).—This is always due to a psychopathic heredity. Patients suffer from weak will power (abulia) and are beset with obsessional or imperative ideas, or are haunted by dreads and fears of doing something they are anxious to avoid.

XI. **Alcoholic Insanity.**—Alcohol is generally accorded the chief place in the causation of toxic insanity. It may result in two special forms : (*a*) delirium tremens, and (*b*) alcoholic dementia ; it may also result in (*c*) delusional and other forms of insanity.

XII. **Syphilitic Insanity.**—(*a*) After contracting, or running the risk of contracting, this loathsome disease it will prey on the minds of some persons to such an extent as to produce an extreme degree of hypochondriasis, to which the term *syphilophobia* is aptly applied. In every symptom, normal or abnormal, they see the disease. The term is also applied to certain patients who have a morbid fear of contracting syphilis, which becomes an obsession with them. (*b*) Syphilitic endarteritis may lead to a dementia indistinguishable from senile dementia except by the age of the patient. Various forms of mania and melancholia may also be associated with arterial and

gummatous lesions, or with concurrent cachexia. (c) General paralysis of the insane is essentially syphilis of the nervous elements (§ 576).

XIII. **Puerperal Insanity** is a generic term for the mental disorder which arises under three different conditions. (a) The mental perversion which arises during *pregnancy* is generally a form of *melancholia*, and varies in degree from a simple exaggeration. of the morbid longings and perverted tastes which are more or less usual during gestation to melancholia of a pronounced type, accompanied, perhaps, by delusions and a suicidal tendency. It is not generally serious unless heredity is in operation, and usually disappears after the confinement. (b) Puerperal *mania* (or puerperal insanity proper) arises usually between the first and fourth week after delivery, coming on usually suddenly with maniacal symptoms. (c) The insanity of *Lactation*, or post-puerperal insanity, is a form of *melancholia* which arises during the first two or three months, or any time during the first twelve months after confinement. All three forms are apt to recur in succeeding pregnancies, but unless there is a hereditary taint of insanity the prognosis is good for recovery. Abundant food and removal from home are indicated, and special measures should be directed to meet the tendency to suicide or infanticide which is frequently present even in the mildest cases, and gives no indication of its presence until some untoward event occurs.

§ 578. **Prognosis and Treatment of insanity in general.**—The *Course* and *Prognosis* in several of the various forms of insanity have been referred to. In general terms the chief points on which the prospect of recovery depends are (1) the absence of heredity, especially direct heredity; (2) the rate of onset of the attack, being more favourable in a rapid than a slow, insidious advent; (3) the duration of the attack before the patient comes under treatment; and (4) the kind of insanity present.

PROSPECT OF RECOVERY IN CHRONIC MENTAL DISORDERS

Good.	Moderate.	Bad.
Hysteria. Alcoholic. Syphilitic (mostly). Puerperal.	Delusional Mania. Melancholia.	G.P.I. Moral insanity. Dementia.

The *Treatment of insanity* in detail has been referred to under the different forms, but the general principles resolve themselves into four indications: (1) feeding; (2) psycho-therapy; (3) placing under restraint; and (4) treatment of insomnia and any physical defect discoverable. Hypnotism (as below) is available for some of the slighter cases, especially where alcoholism is in question.

The question of **removal to an asylum** depends on many things, chiefly (i.) the manageability of the patient; (ii.) the means at home for control; and (iii.) the character of the mental disorder and its potentiality for homicide or suicide.

Any mental patient, however mad, can be taken care of by his or her relations *without certification*, provided it is done without payment or restraint, they being responsible for the patient's safety.

Cases of slight eccentricity and *uncertifiable* mental aberration may be received into the house of a medical man or other householder for payment; but directly a case becomes *certifiable* (in the opinion of the Commissioners) it must be placed under certificate. The penalties for breach of this are very heavy. No medical man or other householder may retain in his house more than one certified patient at a time without special permission from the Commissioners.

A mental patient can be received at a private asylum as a voluntary boarder *without certification* if the patient writes a letter to the Board of Control (66, Victoria Street,

London, S.W) or Visiting Justices stating that he wishes to go there as a voluntary boarder, and his medical attendant reports he is a suitable case. Voluntary boarders may be admitted to Registered Hospitals (e.g., Bethlem) simply by application to the managing committee of the hospital, and this also applies in the case of a special hospital (e g , The Maudsley), but until fresh legislation is enacted patients cannot be received in the rate-paid mental hospitals unless duly certified. A Royal Commission has recently been appointed to consider this matter as well as the provisions of the existing Lunacy Laws.

Procedure for Removal of the Insane under the Lunacy Act.—The procedure for removal is somewhat intricate, and it is usual to remember that the relieving officer of the parish is a most convenient person to apply to, bearing in mind, however, that it is no part of his duty to undertake private cases, but that, nevertheless, if he be approached with due regard to the importance of his office, he may save those concerned a great deal of trouble, and supply them with all the necessary forms and particulars as to modes of procedure. He is also in constant relation with the lunacy justices

A person deemed to be insane, and found *wandering at large* not under proper care, can be apprehended by a " constable, relieving officer, or overseer " of the parish, and taken to the workhouse Any person, either *pauper* or *non pauper*, deemed to be a person of unsound mind can, for his own safety or that of others, be removed from a dwelling house by a relieving officer to the workhouse In either case the patient can be detained there for three days upon the certificate of such constable or relieving officer and, further, upon the certificate of the medical officer of the workhouse, for a total of fourteen days Meantime the procedure under No 3 (c) below can be instituted This method is now often utilised for persons in all classes of life who are dangerous and away from their friends.

In *private cases* the urgency order (1, below) can be used in urgent cases. This holds good for seven days from date of reception ; if not urgent (2) is the usual method.

All the different forms necessary are procurable from Shaw, Fetter Lane, London, or, as previously mentioned, from the relieving officer.

A patient can be removed to an asylum in England or Wales in five ways :
(1) Under an Urgency order signed by a relation (or guardian) and one doctor.
(2) Under a Reception order of a Justice obtained by petition of relative on two doctors' certificates (used also for certification in a case for single care)
(3) Under a Summary Reception order of a Justice
   (a) On information from the police or relieving officer that a non-pauper is cruelly treated or neglected, a Justice calls in two doctors, who certify insanity.
   (b) On information from the police or relieving officer that any person, pauper or not, is wandering at large deemed to be a lunatic, a Justice calls in one doctor who certifies insanity.
   (c) On information from the relieving officer that a pauper is deemed to be a lunatic, a Justice calls in one doctor who certifies insanity.
(4) Under an order after Inquisition, being a written authority from the " Committee " of the person, together with an office copy of the order of the Court of Chancery appointing the " Committee." The " committee " is a legal phrase for the guardian appointed by the court.
(5) Under a Reception order by two Commissioners (rarely done), who call in a doctor who certifies insanity.

The procedure in Ireland and Scotland is somewhat different, as is also that under the Mental Deficiency Act, *vide* p. 823.

Testamentary Capacity.—The intricate technicalities of the lunacy law cannot be entered upon here, but a knowledge of what constitutes the testamentary capacity of a patient is of great importance to the practitioner, because it is often on his evidence that courts of justice decide such matters. The testamentary capacity of a person of unsound mind depends practically on three conditions :

C.M.

1. Did he at the time understand the nature of a will and its effects, and did he understand the extent of the property of which he was disposing ?

2. Did he provide for his relatives, or, if not, why did he leave them out ?

3. Had he any delusion bearing on testamentary matters ?

If these questions can be satisfactorily answered and proven, the will is valid, however eccentric the patient may have been, or even if he was at that time a certified lunatic. The fourth question—undue influence—is a non medical question.

§ 579. **Psycho-therapy.**—Besides the curative effects of change of environment and suitable occupation and recreation, much may be done by the influence of the physician's mind upon that of the patient. This may be attained by :

(a) *Conscious Persuasion*, by means of logical reasoning accompanied by emotional force, and is applicable to some patients who still have " insight " into their condition.

(b) *Suggestion*, which is a process of implanting ideas of a corrective nature ; by this means mental improvement ensues. In some cases a patient may be brought to a relaxed and passive condition in which suggestions are reinforced and this condition is sometimes known as the hypnoidal state

(c) Hypnotism.   (d) Psycho-analysis.

Hypnotism.—Hypnosis may be defined as a condition of partial consciousness resembling sleep, in which the subject's capability to receive and act upon suggestions is greatly increased. This increased suggestibility is made use of by the operator for the implanting of new and healthy conceptions and the removal of morbid ideas, the object being to influence the body through the mind. It is worthy of study, both from a psychological and a medical point of view. It explains many of the cures performed at Lourdes and other shrines, and by faith-healers in all parts of the world.

Bernheim asserts that 80 per cent. of his hospital patients are hypnotisable as far as the stage known as somnambulism ; in his private practice the proportion was much less. Dr. Lloyd Tuckey and the author, in the course of some experiments at Paddington Infirmary, estimated that only about 5 per cent. of the patients there were hypnotisable to that degree. Anæmic young women are perhaps the most easily hypnotised, but strong and healthy men are often susceptible, and it is now established that people in whom there is not the faintest taint of hysteria are subject to hypnotic influence. Wingfield found the undergraduates at Cambridge particularly good subjects, and soldiers and others accustomed to respond to the word of command are very amenable to hypnotic suggestion. Children, too, are good subjects when able to understand what is expected of them, whereas the insane and imbecile are generally unhypnotisable, and hysterical women are by no means the best subjects. It is only by trial one can determine whether a person is hypnotisable.

There are various *methods of hypnotising*. The patient should be put at his ease and seated in a comfortable chair. He is made to relax all his muscles and to fix his gaze on a bright object held about 12 inches above the eyes, so as to cause a slight strain and convergent strabismus. This is the method of " fascination " (Braid), and susceptible persons will fall into a cataleptic or somnambulic condition when submitted to it, without further procedure. In the method of " persuasion," hypnosis is induced by verbal suggestion of sleepy sensations, such as heaviness of the eyelids and limbs, and increasing torpor of mind and body. " Passes," as the mesmerists called them, made by passing the hands, fingers extended, about ¼ inch from the face so as to create a slight draught of air, are often helpful in producing and deepening hypnosis. Whatever method is used, the rationale is the same—it consists of monotonous stimulation of one or more senses, with corresponding inhibition of others, leading to a condition of altered consciousness, in which organic functions as well as mental states become more under the control of the operator. Good therapeutic results are obtainable when only a slight drowsiness is produced, or even indeed when the patient is wholly awake. The patient should be allowed to rest quietly for half an hour, when he should be told to awake. The suggestions should be given in an authoritative and impressive manner, and they be accompanied by manipulation of

the affected part as practised by Braid. They are directed towards the removal of pain, spasm, and other symptoms, and the re-establishment of normal functions. Such suggestions often act immediately, and the action in successful cases is continuous and sustained.

Hypnotism should be used only by medical men, and with proper *precautions*. The consent of the patient and his friends should be obtained, and a third person should be present during the operation. In competent hands no bad effects result from its employment even over prolonged periods, but much evil, moral and physical, might follow the abuse or misuse of this powerful agent. Its use for purposes of public exhibition should be forbidden by law. Hypnotism has been employed to relieve pain, to procure sleep, to cure the effects of grief and shock, to reform alcoholics and moral perverts, to cure various neuroses, such as nocturnal enuresis, and to relieve various hysterical manifestations such as anæsthesia attacks and paralysis. Witterstrand, of Stockholm, claims to have cured many cases of epilepsy by keeping patients in a state of profound hypnosis for three weeks continuously. In exceptional cases hypnotism has been employed as an anæsthetic in surgical and midwifery practice.

Psycho-Analysis.—This is a method of investigating the unconscious mind which has been advocated by Freud. It consists in a minute study of the patient's dreams, and in a process of " free association." The patient is requested to state every thought and word that casually occur to him whilst under examination, in the hope of discovering some hidden psychic trauma of early childhood. Freud emphasises unduly the sexual content of the unconscious mind. He considers that dreams have definite symbolic meaning requiring special interpretation, and that complexes are discovered by their elucidation. Jung has extended the method by " word-associations," using 100 selected stimulus words and observing the character and time of the words of response. Any delay in reaction as shown by a stop-watch indicates that a repressed complex has been affected, which when brought to consciousness and fully explained assists in curing the patient. Psycho-analysis is mostly available in Psychasthenia and Hysteria, but sometimes also in cases of Melancholia. It is of no use in patients past fifty years of age, and its application is limited owing to the time and expense involved—many cases requiring an hour's sitting daily over a period of months. Although possessing therapeutic value in some cases otherwise intractable, psycho-analysis occasionally upsets patients and does harm. It is therefore best left in the hands of experts of acknowledged experience and repute.[1]

§ 580. Mental Deficiency in Adolescence (ten to twenty years) may (1) consist occasionally of one of the chronic mental disorders of adults previously described, and especially the' dementia præcox and MORAL INSANITY described in § 577 ; or (2) it may be a SEQUENCE of either of the two groups below (congenital and non-congenital mental deficiency of childhood) (see § 581). (3) There is also a MENTAL OR MORAL DEFICIENCY inherent in the individual, and manifested for the first time in adolescence, and it is this last class with which we are now concerned ; it includes the " borderland cases," and constitutes at once the puzzle of the alienist and the trial and burden of relatives and guardians:

Symptoms.—Various kinds and degrees of defect may be met with. It is nearly always between the ages of ten and twenty that such cases come under notice for the first time, sometimes because " they will do stupid things," sometimes for " romancing " or lying, sometimes because they do not learn as rapidly as others, or " are not

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[1] The student who desires further information on psycho-analysis is recommended to study Freud's Introductory Lectures (English Translation by Joan Riviere. Geo. Allen & Unwin, Ltd.)

so bright " ; sometimes because they are unruly, or have taken to drink or other vices. In the last-named case, when they have a moral obliquity, they may be possessed of brilliant intellectual gifts, but more often there is an all round deficiency, and they are and remain childish all their lives. This deficiency leads them to consort with all sorts and conditions of men and women, whose habits and language they quickly imitate ;' if sent for a sea voyage, they are generally to be found in the forecastle or steward's pantry, and rapidly assume the indelicate language of the seafaring man. Their parents expostulate with them, and they promise, and do try, to avoid repeating the offences, but they soon break out again. Much trouble accrues to the parents and guardians of such persons to keep them, when belonging to the well to do classes of life, from squandering a fortune, forming an ill judged liaison, or getting into other and worse troubles, and when belonging to the lower classes, to keep them out of prison.

　　Causes —In nearly all such cases there is a neuropathic family history on one or both sides   A few cases can be traced to some of the causes of acquired mental deficiency.   Inbreeding, such as occurs in the aristocracy, may be a contributory cause.   The condition would naturally be aggravated by faulty education and mode of life, but without the hereditary and inherent mental defect it cannot be produced.

　　The *Treatment* is a question of careful training and education, and a good deal can be accomplished if no expense is spared.   It is always best, I am sure, to remove these cases from home, for nearly always a state of friction arises between the girl or youth and one or other parent, which is detrimental to their progress.   If the patient is liable to outbursts of passion, these must be gently curbed, and the system of education made attractive by utilising any particular taste which they have.   The amount of improvement which such cases are capable of depends upon the possibility of fixing the attention, and this depends largely on their having some taste which can be so utilised, such, for instance, as music   The possibility of errors of refraction may be remembered, as in the cases quoted.   The provisions of the Mental Deficiency Act are mentioned on p. 823.

　　§ 581. Mental Defects in Children under Ten are very different to the mental disorders of adults.   Moral perversion, as already mentioned (§ 577), is not infrequent, but mania, melancholia, and other adult varieties are extremely rare.   Mental deficiency as a whole is the charcteristic of this age period, viz , idiocy, imbecility, feeble-mindedness and moral defect. It may be CONGENITAL—*i.e.*, due to factors causing defective germ-plasm —or ACQUIRED (non-congenital)—*i.e.*, due to causes acting upon the fœtus or infant.

　　The *Symptoms* and causes of these two groups differ considerably.   In both varieties the children are " backward," deficient in all the faculties of the mind in greater or less degree ; they carry evidences of this in their manner and behaviour, and some-times, but not always, in their face.   CONGENITAL cases nearly always present some well-marked *alteration in the facial or cranial and bodily conformation*, and very often they are dwarfed in body as well as in mind.   Children belonging to the NON-CONGENI-TAL class of mental deficiency do *not present such marked alterations* ; their expression is bright, their cranium and face natural, their limbs well made, and, excepting in the paralytic class, they can generally walk and run well.   Unfortunately, they are also distinguished from the congenital class by a mobility and restlessness which is an indication of the difficulty of fixing their attention, and therefore of teaching them.

　　The practical point to ascertain in all cases of mental deficiency in childhood is their teachability, and this depends on two questions: (1) Can their attention be attracted and fixed ? and (2) Are they imitative ? (3) Their facial and bodily con-formation should be observed ; (4) the history or evidences of paralysis noted ; and

(5) if they have been to a Council School in England, the standard they have reached —the average in the infants' school is anything under five years; in Standard I., seven to eight years, with an increment of one year for each succeeding standard, Standard VII. being reached by normal children at thirteen or fourteen.

The congenital varieties of mental deficiency in childhood arise from defective germ-plasm owing to neuropathic heredity or alcoholism, tubercle, or syphilis in the parents. The children generally present evidences in the facial and cranial conformation as well as in the mind. In a good many of these cases the palate is too high, or V-shaped, too narrow from side to side, or otherwise deformed. The palate described by Sir T. Clouston, and called by him the "neurotic palate," is one midway between the normal palate and the V-shaped palate, and is found in persons of nervous temperament, who are liable to hysteria, neuralgia, and migraine. Dr. Fletcher Beach found that out of 700 feeble-minded children 28 per cent. had V-shaped or otherwise deformed palates, and 60 per cent. had neurotic palates. On the other hand, a high or V-shaped palate does not necessarily imply congenital deficiency, as was at first thought. The acquired varieties occur from factors arising before, during, or after birth—e.g., trauma, malnutrition, infectious fevers, etc.

The VARIETIES OF CONGENITAL IDIOCY are three in number:

1. *Simple or Genetous Idiocy* includes children *without any obvious* abnormality of the cranium or limbs, only in the face or palate (*supra*). In some the facial expression may be fairly intelligent, but most of the lower grade present an animal expression, thick lips, pug-nose, large coarse ears, broad, thick, depressed bridge of nose, narrow or hairy forehead, and underhung jaw.

2. The *Mongol* or *Chinese* type of congenital deficiency is so called from the resemblance of the face to that of the Chinese, the palpebral fissures sloping downwards and inwards. With flat face, flat back to the head, and constant protrusions of the tongue, this form of idiocy presents an unmistakable physiognomy. The fingers also are stunted and the little fingers incurved. Congenital heart disease occurs in about 30 per cent. They may be regarded as "unfinished" children, as they are often born of mothers who have suffered from continued ill-health during pregnancy; sometimes they are the youngest of a large family, or born of parents advanced in life. These children are imitative, and therefore educable to a limited extent, but they make no progress beyond a certain point.

3. *Microcephalic* idiocy includes children whose heads are smaller in circumference than normal, which averages about 19 inches. The head may measure 17, 15, or even 12 inches; the forehead is narrow, and slopes backwards, corresponding with the deficiency of the frontal development of the brain. The features are frequently normal, eyes large, and nose aquiline. These children rarely make much improvement, for they have but little power of attention, though some of them are imitative. The question of craniectomy was raised some years ago in connection with these cases, as the small size of the brain was thought to be due to premature union of the cranial sutures, but this is now known to be incorrect.

Causes of CONGENITAL MENTAL DEFICIENCY in children.—Causes acting *in the parents before birth* (heredity) occupy a very prominent place. Beach and Shuttleworth have exhaustively investigated this question in 2,400 cases. Abnormal conditions in the mother during pregnancy account for as much as 29 per cent., among which may be mentioned injuries, worry, anxiety, fright, or illness. Phthisis in the parents accounts for 28 per cent., insanity and imbecility 21 per cent., epilepsy and other neuroses 20 per cent., and intemperance in the parents, 16 per cent. Consanguinity acted as a cause in only 4 per cent., even when consanguinity in the grandparents was taken into account. This is somewhat at variance with the generally supposed evil of marriage between blood relations, but it is a fact about which there seems no doubt, and Dr. Beach adds: "Even in those cases where consanguinity was present there were other hereditary tendencies, of themselves quite sufficient to produce the affection (congenital mental deficiency) without the presence of consanguinity." Syphilis in the parents was responsible for only 1% the effect of syphilis in the parents

apparently being not so much the procreation of idiots as of children apparently healthy at birth whose nervous systems break down at the age of puberty.

Acquired (or non-congenital) mental deficiency in children under ten is rarely difficult to differentiate from the congenital deficiency (*vide supra*). Among the *Causes* infantile convulsions were in operation in 27 per cent. of the 2,400 cases which Dr. Beach collected, epilepsy and other cerebral affections in 8 per cent., head injury in 6 per cent., fright or shock in 3 per cent., febrile diseases such as scarlatina, measles, whooping-cough, enteric, small-pox in 6 per cent., and over-pressure at school was only mentioned in 0 16 per cent. Among the causes acting *at the time of birth* prolonged or tedious parturition is undoubtedly the most potent. It is sometimes supposed that injury by forceps is a potent cause of the production of congenital mental deficiency, but it is worthy of note that in only 3 per cent. of Beach and Shuttleworth's cases was there a history of delivery by forceps. His facts go to show that it is far better to put on the forceps early than to allow labour to be prolonged indefinitely. Such prolongation leads to great compression of the cranium, the brain being crushed, distorted, and otherwise injured. Such children when born are very often in an absolutely helpless condition. Others are subject to convulsions, and when death ensues, meningeal hæmorrhage and cortical laceration are very often found.

The **varieties of Acquired Idiocy** are nine in number :

1. *Hydrocephalic*, due to occlusion of the foramina of Majendie or Munro causing distention of fluid and atrophy of the cortex. Most cases are quiet and docile.

2. *Hypertrophic*, from overgrowth of neuroglia either in the diffuse or nodular form. It is somewhat rare and occasionally has a skin eruption associated with it (adenoma sebaceum).

3. *Eclampsic.*—Infantile convulsions, if only occurring once or twice, may have no evil result, but if continued for some time throughout the early months of life generally leave the child feeble-minded. Many such cases occur in the children of insane or epileptic parents.

4. *Epileptic.*—As a rule epilepsy does not commence until after ten years of age, but where it starts before the age of seven mental defect results. Infantile convulsions cease before two years and epilepsy never occurs till after two.

5. *Paralytic.*—This is associated with hemiplegia or diplegia, the result of cerebral hæmorrhage, from trauma at birth and occasionally from a fall during early life. Sometimes these cases are associated with spasticity or choreiform movements and symptoms are produced due to a cerebral scar.

6. *Inflammatory.*—This follows from encephalitis and meningitis, from scarlet fever or other exanthema and the mental defect may not supervene till later, which is also prevalent in endemic encephalitis.

7. *Syphilitic.*—Signs of congenital syphilis are often present, and the mental defect is exhibited about the time of the eruption of the permanent teeth.

8. *Idiocy from Deprivation of the Senses.*—The mind is cut off from environmental stimuli owing to sight and hearing being affected from acute infections, trauma or hæmorrhage. The defect may be remedied by means of special training.

9. *Cretinism.*—This may be endemic or sporadic. The head is usually large, flat at the top, spread out at the sides (Fig. 6, § 19). Hair coarse and dry and voice squeaky. Under treatment by thyroid gland or extract these cases make remarkable progress as in Figures, but the treatment must be continued during the whole of life.

The *Prognosis* of MENTAL DEFICIENCY in childhood is always grave. Many succumb early and there are few cases where much improvement takes place when once the deficiency is established. If the mental symptoms seem to have come to a standstill and the child is in any degree educable, something may be done in this direction ; but, as a rule, less can be done for acquired cases than for congenital, and their tendency is to get worse.

In the TREATMENT OF MENTAL DEFICIENCY IN CHILDREN a certain amount can be done by education and discipline in Special Schools for Defectives. The first and fundamental principle is to remove the child from the home and from the surrounding

conditions which have been unconsciously adapted to the defective habits. The second point is to fix the child's attention, and this can best be done by employing music, pictures, and other things which please and attract the child. It may be taken as a general principle that the educability of a child—that is to say, the prospect of recovery—depends entirely upon the facility with which the attention can be attracted and the efficiency with which it can be retained when once secured. The shape of the head and aspect of the face cannot be relied upon; the brightest looking children are often the most volatile. On the other hand, the dull-looking children who take an interest in their work will make more progress. As a rule, the prognosis is much worse in irritable, restless cases than in quiet cases. It is usual to commence with the education of the senses—touch first, then sight, hearing, taste, and smell. Speech also has to be educated by means of speech drill. Any vicious habits must be gently corrected, and punctuality and discipline enforced. Later on, perseverance, and ideas of justice, duty, self-reliance, prudence, and forethought have to be inculcated. The progress of any case under treatment depends very much on the age at which it is commenced. It should always be begun as early as possible, and the idea which some mothers have of the symptoms passing off at seven or fourteen by an abrupt change from mental enfeeblement to mental brightness should be met and combated as wholly-fallacious, and very damaging to the prospects of success.

The *Mental Deficiency Act* is devised to protect and control defectives, and thus to prevent them from doing harm to themselves and to society. Hitherto on leaving the special schools no further supervision was possible for most of them. Mild cases of defect can be received in approved homes without certification. Other cases can be placed under single care, or in houses and institutions certified under the Act, including state institutions. Two medical certificates are necessary for all grades, and for an idiot or imbecile the order of the parent or guardian suffices, but for the feeble-minded and moral defectives the intervention of a Justice is required in addition. The requisite forms for the Act may be procured from Shaw, Fetter Lane, London.

GROUP III. INTRACRANIAL INFLAMMATION

§ 582. Pyrexial disorders of the nervous system are not numerous, and are practically confined—if acute anterior poliomyelitis and certain exceptional cases of cerebral abscess and cerebral hæmorrhage be omitted—to acute inflammation of the meninges. They consist of the following intracranial inflammatory conditions, all of which present *cerebral symptoms with pyrexia* and its attendant symptoms. The onset in most is acute.

I. Tuberculous meningitis (§ 583).
II. Acute meningitis (§ 584).
III. Post-basic meningitis (§ 584).
IV. Epidemic cerebro-spinal meningitis (§ 401).
V. Intracranial abscess (§ 586).
VI. Sinus thrombosis (especially septic thrombosis) (§ 586).
VII. Hæmorrhage, chronic degenerative diseases, etc.
VIII. Encephalitis lethargica (§ 593).

CLINICAL INVESTIGATION.—1. The *cerebral symptoms* in intracranial inflammation may be grouped into those of irritation and compression. The symptoms of cortical *irritation* which are the most usual, especially in the early stages, consists of headache, vomiting, tonic or clonic spasms or convulsions, sleeplessness, restlessness, delirium, quick pulse, and contracted pupils. The symptoms of *compression*, which usually ensue later, are mental dullness, paralysis of the limbs and cranial nerves, a slow full pulse, dilated or unequal pupils, and stupor passing on to coma. The earlier irritative stages may have to be diagnosed from other causes of convulsions (§ 634); the later compression stage from the typhoid state (§ 373) or coma (§ 564).

2. The *scalp and cranial bones* should be examined for any swelling or tenderness on percussion—*e.g.*, any œdema (sinus thrombosis), erysipelas or Pott's puffy tumour (an œdematous swelling of the scalp which is usually a sign of subcranial extra-dural abscess in that situation). When there has been a *wound of the scalp*, the skin may have healed up externally though pus has formed beneath, and the infective products carried along the perivascular sheaths or lymphatics into the skull; but the patient may go about for days or weeks before symptoms of intracranial inflammation present themselves.

3. The *ear* and venous *sinuses* near it, the *nose* and adjacent sinuses, notably the frontal sinuses (by means of transillumination), and the *pharynx* should also be carefully examined for any discharge or other signs of disease.

4. The *cranial nerves* need to be examined severally (§ 645 *et seq.*). Inflammation, which, like tuberculous meningitis, has a predilection for the base, nearly always produces cranial paralysis at some time; when it affects the convexity of the brain, it tends to cause convulsions.

5. The *lungs* and other organs should be examined. Many cases of tuberculous meningitis are secondary to tuberculosis of the lungs or peritoneum.

6. The *age* and *history* of the patient, especially as regards any previous otorrhœa, may be noted. Acute meningitis may occur at any age, and runs a course of about two days to two weeks; tuberculous meningitis chiefly affects children, and runs a course of two weeks to two months (including premonitory stage); post-basic meningitis is almost confined to infants under one year, and runs a prolonged and indefinite course.

7. The *temperature* should be carefully investigated, and a chart obtained. Diurnal intermissions suggest tuberculous meningitis (§ 583). A nondescript or continued temperature suggests acute meningitis (§ 584). The temperature falling after a few days of initial rise and becoming subnormal suggests cerebral abscess (§ 585). Wide and irregular intermissions suggest septic thrombosis (§ 586).

8. Lumbar puncture is a valuable aid both to diagnosis (pp. 952 and 956) and treatment (§ 666).

§ 583. I. Tuberculous Meningitis (tuberculosis of the cerebral meninges) is the commonest form of intracranial inflammation in children. The cerebral pia mater, especially at the base and in the Sylvian fissure, becomes studded with grey miliary tubercles. The disease occurs chiefly in children, and in the male sex. It may (i.) supervene in a case of tuberculosis elsewhere, of which symptoms have existed previously; it may (ii.) be ushered in suddenly with vomiting and convulsions in a child previously in good health; or it may (iii.) come on insidiously as a primary affection of the meninges, with a long prodromal period.

The *Symptoms* are usually insidious and variable. A prodromal stage is followed by three other stages, the latter running a course of two or three weeks, and passing imperceptibly into each other. In the *prodromal* stage, which may last a considerable time, the child gets thinner, loses appetite, is peevish and listless but with intervals of brightness, when the mother thinks it is getting well again. Intermittent pyrexia may be revealed from time to time. (1) The *irritative* stage is ushered in, very often suddenly, with (i.) severe headache, vomiting, or convulsions, and there may be delirium, or the child may lie curled up, shrinking from light or sound. (ii.) There is a moderate degree of fever, usually of a diurnally intermittent type. (iii.) The pulse is quick, and the pupils are contracted. (iv.) The head generally shows a characteristic retraction with rigidity

of the muscles of the neck. (v.) The abdomen is retracted. When vomiting without diarrhœa is present in a child, tuberculous meningitis, or other head mischief should be suspected. (vi.) The peculiar cry, known as the "hydrocephalic cry," may accompany this stage of the disease and last till the end. As a rule the irritative stage lasts only a few days to a week. (2) The stage of *compression* is announced by symptoms pointing to paralysis of the cranial nerves as in all diseases situated at the base of the skull. (i.) The pupils are unequal, and become dilated, and strabismus is common owing to paralysis of the sixth or third nerves. (ii.) The patient is drowsy, with slow pulse, occasionally altering in rate, and sighing respiration. (iii.) There is vaso-motor paralysis, indicated by the red streak following the track of the finger-nail drawn along the skin. This condition is known as the "tâche cérébrale" (Trousseau), from the idea, now known to be mistaken, that it is found only in cerebral lesions. (iv.) Optic neuritis or tubercle of the choroid may develop. (3) The *final* stage is marked by increasing irregularity and weakness of the pulse and the respiration. The drowsiness passes into coma, and there is incontinence of urine and fæces. Mucus gathers in the bronchial tubes, and breathing is laboured, often taking the Cheyne-Stokes form. Just at the end the temperature may rapidly fall, or it may rise very high (106° or 107° F.), and local paralyses—*e.g.*, of the arm or leg—or ptosis may occur with convulsions. This stage lasts only a few days.

Tuberculous meningitis in adults is often secondary to tubercle in the lungs, abdomen, or elsewhere. It differs from the disease in children in : (1) The advent is most insidious and prolonged. I once saw a typical example of this where headache and intermitting pyrexia were the only symptoms for nine weeks. (2) Severe and persistent headache and intermitting pyrexia are always prominent features. Hemiplegia, or, rather, hemiparesis, may be the earliest and principal symptom, and aphasia is not infrequent ; while (3) optic neuritis, strabismus, or other evidence of cranial nerve paralysis, such as facial paresis, or inability to swallow, are present as in children. Vomiting and convulsions are rare. (4) The course lasts from five to twelve or more weeks.

The *Diagnosis* of tuberculous meningitis may often be settled by lumbar puncture (pp. 952 and 956, in § 666). *Enteric fever*, with head symptoms, and no rash or diarrhœa, may for a week or longer be mistaken for meningitis ; and, on the other hand, meningitis in which there is little headache may be mistaken for enteric fever. But the course of the disease and the absence of the Widal reaction soon reveal its true nature. Any one of the specific fevers may be ushered in with headache so severe as to give rise to suspicion of meningitis ; but in all such cases the headache ceases when delirium begins. Irregularity in the breathing and the pulse, and evidences of basal paralyses are conclusive signs of meningitis. *Acute meningitis* is distinguished from the tuberculous variety by its rapid course, which rarely exceeds a few days, by there being no basal paralyses, and by the presence, perhaps, of a local cause, such as injury or otitis media. A rapidly-growing *tumour—e.g.*, tuberculous or gliomatous—may simulate

meningitis. Here the character of the optic neuritis present aids diagnosis—if very intense, with swelling and hæmorrhages, it is probably due to tumour. The early stages of tuberculous meningitis in young women may resemble *hysteria* : the eyes should be examined for optic neuritis. *Postbasic mёningitis* occurs in infants under one year, and has a longer course. In *marasmic conditions* in children drowsiness and convulsions may arouse the suspicion of tuberculous disease being present, but in such cases the fontanelle is depressed from a lowering of the intracranial pressure.

Prognosis and Treatment.—Tuberculous meningitis is essentially a sub, acute disorder. Its average duration varies considerably—from three weeks to three months are about the limits. The prognosis depends mainly on three things—first, the period of the disease at which the patient comes under treatment ; secondly, the height and range of the temperature, which is the measure of the activity of all tuberculous processes ; and, thirdly, the extent of the tuberculous mischief in other organs. Until recent years it was regarded as necessarily fatal, but cases of undoubted recovery have been published. Curiously enough, the *treatment* which was most successful consisted of calomel and pot. iod., remedies which are directed against syphilitic lesions, and it seems possible that some of the alleged cases of recovery may have been syphilitic meningitis. Lumbar puncture (§ 666) has in several instances resulted in recovery. 10 or 20 c.c. may be withdrawn and the operation may be repeated if necessary. Ice-bags applied to the head, bromides or chloral internally, or evaporating lotions to the shaven scalp, relieve the headache. Mercurial and iodoform ointment rubbed into the scalp is recommended in chronic cases.

§ 584. II. **Acute Meningitis** (Acute Hydrocephalus) is a diffuse inflammation of the pia mater and arachnoid (leptomeningitis), simple or purulent, according to its exciting cause, chiefly affecting the convexity of the brain.

Symptoms.—There are symptoms and signs of cortical *irritation* (§ 582), followed by signs of *compression*—i.e., increased intracranial pressure. The inflammation affects the convexity rather than the base of the brain, the reverse of tuberculous meningitis, and the symptoms, therefore, consist of muscular twitchings, spasms, sometimes convulsions, and later on paralysis of the muscles of the body, rather than paralysis of the cranial nerves as in tubercular meningitis. There is no prodromal stage, the invasion being sudden. The disease lasts usually a few days to one or two weeks. (1) The temperature runs a pyæmic course with wide variations, and there may be rigors. Other symptoms are (2) severe persistent headache ; (3) delirium ; (4) retraction of the head, rigidity of the muscles of the neck, diffuse hyperæsthesia and pain, especially about the neck, from involvement of the spinal nerve roots. Optic neuritis and ocular paresis are rare. (5) There may also be vomiting, flushing of the face and conjunctivæ, the "tâche cérébrale " of Trousseau, and herpes on the lips or face. Kernig's sign (§ 547) is present if the inflammation extends to the spine. There is usually a history or evidence of some cause, since the disease is rarely primary, the most common cause being disease of the petrous bone with or without ear disease, attended by a purulent discharge.

Etiology.—The most frequent cause of purulent meningitis is (1) extension of inflammation from adjacent parts, either from *without*—e.g., in caries or necrosis of the cranial bones, erysipelas of the face or scalp—or from *within*—e.g. a cerebral abscess or sinus thrombosis. Any of the bones may be involved, but disease of the petrous bone is the most frequent cause, with or without otitis media : frontal sinus

or ethmoid disease is a much less common cause. In disease of the cranial bones a chronic localised thickening of the meninges may prevent a localised collection of pus (a subcranial abscess) from spreading laterally ; but the edges are liable at any time to become softened, and so to permit of a generalised purulent meningitis. (2) Punctured wounds of the scalp may cause meningitis in the same way as in cerebral abscess formation (*q.v.*). (3) Pyæmia and malignant endocarditis may be attended by a purulent, and rapidly fatal form of the disease. (4) The pneumococcus is probably one of the commonest causes of suppurative meningitis. A *primary* pneumococcal meningitis may occur without pneumonia being present. (5) It may occur secondarily to other constitutional disorders such as the specific fevers, influenza, diphtheria, acute rheumatism, measles, small-pox, erysipelas, scarlet fever, anthrax, gonorrhœa, and actinomycosis, also, it is said, to gout, chronic nephritis, and heart disease in the terminal stages. (6) A primary cerebro-spinal meningitis also occurs in the sporadic and epidemic form, due to the diplococcus intracellularis (§ 667). (7) *Tuberculous* and *post-basic* meningitis are special forms described respectively in § 583 and below.

Diagnosis.—Acute meningitis has to be diagnosed from other forms of meningitis and from *acute specific fevers*, pneumonia, and other acute diseases, which come on with headache and vomiting. *Pneumonia* may be very deceptive at first before the lung signs are definite, but the rapidity of respiration affords us a clue. *Tuberculous* meningitis has a more insidious onset, a prodromal stage and a prolonged course. When there is otitis media, meningitis may require to be differentiated from *cerebral abscess* ; this has more localised symptoms, a low temperature after the first day or so, and the headache lasts longer before coma sets in. In septic *sinus thrombosis* the temperature is marked with recurring rigors, and local signs of the sinus involved are usually present, such as the brawny swelling in the neck in lateral sinus thrombosis. The diagnosis of the *cause* also requires investigation, especially in children. When due to otitis, the patient usually holds the hand to the head. Lumbar puncture (§ 666) and cultures may be helpful here.

Prognosis.—The disease usually runs a fatal course in a few days, but it may completely be recovered from, or if traumatic in origin, it may pass into a chronic localised meningitis. Recovery has been reported after the primary pneumococcal form, though this and the other septic forms are generally fatal.

Treatment.—The patient must be kept in a dark room, perfectly quiet, the head shaved, and an ice-bag applied. A purge should be given, preferably of calomel. Only milk diet is allowed. Mercury, especially in the form of inunction, is recommended, together with large doses of iodide of potassium. The cause must be carefully investigated, and where local causes are in operation, such as injury or disease of the scalp, these must be treated surgically. In children and infants especially, puncture of the tympanum, with consequent outlet of a little pus, has often resulted in the prompt relief of symptoms of intracranial irritation. Lumbar puncture (§ 666) not only affords a clue to diagnosis of the primary malady, but may be useful by the relief of tension.

III. **Posterior Basic Infantile Meningitis** may be acute, but is usually chronic. It occurs in infants under twelve months, and is due to inflammation in the posterior fossa of the skull, associated with a special diplococcus, the diplococcus of Weichselbaum. This is the same microbe as that of epidemic cerebro-spinal meningitis (§ 401), and post-basic meningitis is now generally regarded as a sporadic, sub-acute form of that disease. The exudation glues together the cerebellum and medulla, leading ultimately to the blocking of the foramen of Majendie and distension of the ventricles (hydrocephalus). The characteristic *Symptoms* are (1) the gradual onset of the retraction of the head, which may amount to opisthotonos of the spine with flexor and extensor spasm of the limbs ; (2) staring of the eyes, with blindness, appears quite early in the disease, unassociated with optic neuritis, being due to involvement of the occipital cortex ; (3) vomiting ; (4) rigidity of the limbs, which may be general or localised to one extremity ; (5) paroxysms of high fever lasting a day or two. The

disease occurs in infants from three to twelve months old, only occasionally in older children. It is diagnosed from tuberculous meningitis by the age of the patient, the greater degree of cervical opisthotonos, the longer course of the disease, the absence of optic neuritis ; and by lumbar puncture.

Prognosis.—The disease often runs a prolonged course of weeks or months. Death has taken place after nineteen months. Cases may undoubtedly recover ; Drs. Lees and Barlow say one in six recovers completely. In others grave sequelæ remain : hydrocephalus resulting from the distension of the ventricles ; blindness (without optic neuritis) ; deafness (with consequent loss of speech) ; and defective intelligence ; or a combination of these.

Treatment.—Mercurial ointment should be rubbed into the neck, and iodide of potassium in doses of 1 to 3 grains every two hours is believed to cause absorption of the exudation. Lumbar puncture affords both a clue to *diagnosis* (§ 666) and a means of *treatment.*

IV. **Epidemic Cerebro-Spinal Meningitis** has already been described in § 401.

§ 585. V. **Intracranial Abscess.**—Abscess of the brain may occur in an acute or chronic form.

The symptoms of cold or *chronic abscess* are identical with those of any other cerebral tumour, apart from the history and the symptoms referable to the condition which caused it. The commonest cause of cerebral abscess is middle-ear disease, but in quite a number of cases there is no history of any cause, and these are indistinguishable from tumour.

The course of acute abscess of the brain may be divided into three stages... The *initial* or inflammatory stage lasts from twelve hours to three days, or longer. Very often this stage may not be noticed ; and in most instances the patient does not come under observation until it is past. The three chief symptoms which characterise this stage are *pain, vomiting, and rigors.* (i.) The pain in the head may be burning, shooting, continuous or intermittent, and there is tenderness over the seat of the abscess. (ii.) The vomiting occurs without nausea, and has no relation to food. (iii.) The rigors vary greatly in severity, from a slight shivering to a shuddering. If they are frequent, some systemic infection or sinus thrombosis is probably present (*q.v.*). The temperature in this stage is above normal, but not high. If the disease be due to otitis media the discharge stops. The *second or collapse* stage shows symptoms similar to the first stage, only much less marked. (i.) The pain is diminished ; the patient may lie quietly moaning with the hand over the affected part. Tenderness is brought out on percussion, and is useful in localising the site of the mischief. (ii.) Vomiting and vertigo occur on movement only. (iii.) Cerebration is slow ; the patient will answer questions correctly, but only after long delay, and he may fall asleep in the middle of a sentence. (iv.) The temperature in this stage is normal or subnormal, if the abscess is uncomplicated. (v.) The pulse is slow and full (30 to 60). Respiration is slowed. (vi.) Optic neuritis develops ; and (vii.) paralyses may occur, which aid in diagnosing the site of the lesion. This stage lasts from one to five weeks. In the *third* or paralytic stage the abscess terminates in one of three ways : (i.) By compression symptoms—deepening stupor, coma, and death. (ii.) The abscess may open on the surface of the brain, leading to meningeal symptoms with high temperature, quick pulse, vomiting, and convulsions. (iii.) The abscess may open into the ventricles of the brain, an event characterised by lividity, dilatation of the pupils, stertorous breathing, high temperature, convulsions, coma, and death within twelve hours.

Diagnosis.—In *acute meningitis* the symptoms are more of an irritative character, with high temperature and quickened pulse ; lumbar puncture will reveal a leucocytosis. In *sinus thrombosis* there are recurring rigors and local signs of the involvement of the sinus. Both meningitis and thrombosis may accompany abscess, and in this case the symptoms are confusing. If meningitis and abscess occur together, the presence of the latter will be indicated by a slower pulse than would be met with in uncomplicated meningitis. *Intracranial tumours* resemble abscess. The slow progress of the symptoms, the greater amount of optic neuritis, the more definite focal

phenomena, the absence of rigors, and of any source of abscess formation, are in favour of a diagnosis of tumour; while the presence of a subnormal temperature is in favour of abscess. In cases of abscess leucocytosis is present in the blood.

' *Causes* of cerebral abscess.—The chief causes are : 1. *Middle-ear disease* is by far the most common cause. The abscess occurs in the temporo-sphenoidal lobe, or less commonly in the cerebellum. There is usually necrosis or tuberculous disease (caries) of the petrous bone. (i.) The inflammatory process spreads to the meninges, and if local adhesions be present preventing lateral extension, the process goes on, even without any erosion of the bone, to ulceration of the brain, and consequent pus formation within the brain substance. (ii.) Or cerebral abscess may form in the white matter, without implication of the grey cortex. In this case the inflammation spreads along the perivascular sheaths of the vessels or along the veins entering the brain. In short, abscess may occur (*a*) by contiguity or (*b*) by extension from a distance along the lymphatic sheath of the vessels, or along the veins. 2. *Disease of the frontal sinus, of the antrum of Highmore or nasal fossæ*, causes abscess in the frontal lobe. 3. *Injury, such as compound fracture*, with necrosis of the bone, may cause an abscess. 4. *Wounds of the scalp* may cause subdural abscess by the conveyance of septic organisms through the bone. 5. *Carbuncle of the face* (rare) or *scalp* causes abscess, by the spreading of the inflammation along the facial vein and pterygoid plexus, or the ophthalmic vein, to the cavernous sinus. 6. *Disease of the orbit* causes abscess by a similar method of extension. 7. *Erysipelas* (rare) more often causes leptomeningitis. 8. *Abscess of pyæmic origin* may occur in any part, but most commonly in the occipital lobe. The pyæmic sources are septic embolism—*e.g.*, in malignant endocarditis—and pyæmia. 9. The dysenteric amœba is a cause rarely suspected in this country.

Prognosis and treatment.—Early diagnosis and correct localisation are most important, for surgical treatment is successful, but untreated cases usually die. In some cases the abscess undergoes apparent cure, and, if small, may remain encapsuled for years, or even dry up. Sir W. Macewen mentions a few cases where the abscess discharged externally through eroded bone. The usual course of an acute uncomplicated abscess is from two to six weeks. A chronic abscess may last months or years, but is always a source of danger. After remaining without symptoms for years, it may suddenly burst into the meninges or ventricles. Uncomplicated abscess is readily amenable to *treatment* in the hands of a skilled surgeon, but if symptoms of meningitis or sinus thrombosis accompany it, the prospect is much more grave, especially if the meningitis is diffuse. The dysenteric abscess yields to emetine.

§ 586. VI. **Sinus Thrombosis**, thrombosis of the cerebral sinuses, may be PYOGENIC, and accompanied by pyrexia and stupor, or NON-PYOGENIC, accompanied by stupor only.

(*a*) In **Pyogenic Sinus Thrombosis** there is pyrexia of a pyæmic type—*i.e.*, wide variations with rigors. Any one of the sinuses may be involved, but the lateral sinus (secondary to suppurative middle-ear disease) is the favourite position.

The *Symptoms* of septic sinus thrombosis are (i.) severe headache, vomiting, and high fever of a pyogenic type, accompanied by rigors and sweats (see chart, § 413); (ii.) optic neuritis supervening in a day or two, and perhaps photophobia; (iii.) drowsiness deepening into coma, and if operative measures be not prompt, ending in death. In *lateral sinus* thrombosis there are pain and tenderness in the mastoid region, together with the other signs of a suppurative otitis media ; the inflammation spreads down the jugular vein on the same side and backwards behind the mastoid ; and consequently there is generally some hard brawny swelling in these positions ; if there has previously been a discharge from the ear, it usually ceases. When the *longitudinal sinus* is thrombosed, the localising signs consist of œdema of the scalp, distension of the veins over the forehead, and sometimes strabismus, associated with convulsions at the onset. This is the sinus which is most often affected by non-pyogenic thrombosis ; when affected with pyogenic thrombosis the cause is usually some septic lesion of the face or scalp. When the *cavernous sinus* is affected, the localising signs are œdema of the eyelids and root of the nose, sometimes also of the pharynx, exophthal-

mos, and paralysis of the second, third, fourth, ophthalmic division of the fifth and sixth nerves. Pyogenic thrombosis of this sinus may arise from some septic lesion of the orbit, nose, pharynx, or face. The *Diagnosis* of pyogenic sinus thrombosis from *septicæmia* is very difficult, unless the local signs are pronounced. In *acute meningitis* there is a lower temperature without rigors and sweats, and there may be retraction of the head and neck. In *cerebral abscess* the temperature is normal or subnormal after the initial rise, and it is accompanied not so much by stupor as by headache and paralysis of the cranial nerves. The *Prognosis* of pyogenic sinus thrombosis is exceedingly grave, unless promptly dealt with by surgical measures, such as trephining over the lateral sinus, turning out the clot, tying the jugular vein below, cutting down upon the mastoid cells.

(*b*) Non-Pyogenic conditions. It occurs in Sinus Thrombosis from prolonged diarrhœa, the aged, and in adults in the last stages of exhausting diseases, such as phthisis, or cancer, and in chlorosis. The superior longitudinal is the sinus most often affected. Headache, drowsiness, and coma are the leading symptoms; the temperature may be slightly raised, but it is never a prominent feature; in adults delirium may mark the onset. The condition is difficult to diagnose with certainty. In infants the coma supervenes almost imperceptibly upon the drowsiness which accompanies exhaustion. In children epistaxis and convulsions should make one suspect the condition, even in the absence of localising signs. The localising signs are mentioned under Septic Sinus Thrombosis. The *Prognosis* is grave, especially in adults. Children may recover, but at the expense of impaired intellect.

Treatment.—The patient should be kept at rest, with the head and shoulders slightly raised. The neck must not be bent, lest the blood be hindered in its return. Tonics and stimulants may be given.

VII. **Pyrexia**, usually of short duration, may accompany certain intracranial lesions of acute onset, such as (i.) hæmorrhage, especially into the pons, when there is sudden coma, contracted pupils, and hyperpyrexia (see Apoplexy, § 565); (ii.) lesions occurring in the course of chronic degenerative disease of the central nervous system, such as disseminated sclerosis and G.P.I.

§ 587. VIII. Encephalitis Lethargica.—The recent epidemic broke out in Vienna in 1917, and first appeared in England in the spring of 1918. It often begins with symptoms which are referred to influenza. Among the early symptoms are fever, somnolence, headache, double vision and lassitude. Double vision is very common. The virus picks out the extra-pyramidal motor tract at the base of the brain, causing the characteristic Parkinsonian rigidity. (There is some doubt whether, as has been thought, the lenticular nucleus of the corpus striatum and the locus niger are affected. The ocular nuclei are commonly affected, but the nuclei of other cranial nerves may be involved giving rise to paralysis of the face, tongue, and soft palate.) The disease is highly protean in its manifestations. Somnolence and mild rigidity are prominent features. In severe cases the patient is like a waxen image, though he can be roused to a struggle, and refuse when asked to put out his tongue, whereupon he lapses again into stupor. A curious feature is the tendency to cataleptic rigidity; if, when the patient's eyes are closed, a limb is put in a given position, it tends to maintain that position. Variations in type occur, choreiform movements, sudden shock-like spasms, Jacksonian fits, athetosis and symptomatic paralysis agitans. Of 500 cases notified in 1919, exactly half proved fatal, but inasmuch as many mild cases escape detection, the actual mortality is considerably less.

Professor McIntosh, using a filtered emulsion of cerebral tissue obtained from a fatal case, has succeeded in transmitting the disease to a monkey, and from this monkey through a series of monkeys and rabbits. The virus is filtrable and apparently enters through the naso-pharynx. As in the case of syphilis and disseminated sclerosis, it appears to retain its activity for years in the host, often giving rise to a slowly progressive disease. The disease occurs at all ages, with a preference for middle life and affects both sexes. It is but little infectious, most of the cases being isolated

or sporadic. Nevertheless it is advisable for those who are brought in contact with it to use a nasal spray or douche, and to gargle the throat with an antiseptic solution. There is no specific treatment.

GROUP IV. MOTOR DISORDERS OF THE NERVOUS SYSTEM

Motor disorders of the nervous system may consist of PARALYSIS (*below*), INCO-ORDINATION (§ 613), OVER ACTION (§ 618), or MUSCULAR ATROPHY (§ 637).

• *a*. PARALYSIS

Paralysis may take the form of --

Hemiplegia	..	..	..	..	..	..	..	..	..	§ 588
Paraplegia	..	..	..	..	..	..	..	..	..	§ 592
Brachiplegia	..	..	..	..	..	..	..	..	..	§ 603
Monoplegia and single nerve paralysis	..	..	.		..	..	§ 604
Generalised paralysis	..	..	..	..	..	..	..	..	§ 609

§ 588. **Hemiplegia,** paralysis of one side of the body, is due to a one-sided lesion of the motor tract somewhere above the decussation in the medulla. The usual situation is the internal capsule.

Symptoms.—The loss of power in the limbs is almost always on the same side as that in the face ; when it is on opposite sides it is known as " crossed hemiplegia," and the lesion is situated in the pons. The arm is usually more affected than the leg, and the leg than the face. Only the lower part of the face is affected, the food collects round the teeth on that side, but the patient can close both eyes. If the tongue is paralysed, it is turned to the affected side during protrusion, owing to the unbalanced action of the healthy genio-hyoid and genio-hyoglossus. Some hemian-æsthesia on the paralysed side, especially of the hand, is often present from involvement of the hinder limb of the internal capsule. There may be an initial or *early* rigidity at the outset lasting a few hours (as in cases of hæmorrhage), or coming in a few days' time and lasting for a few weeks (as in cases of irritative lesion). There is also a *late* rigidity, due to descending lateral sclerosis,[1] which comes on in nearly all cases of hemiplegia after a few weeks or months, and is permanent. In the course of years the least paralysed muscles may undergo *contracture* from unopposed action, and they are also liable to various kinds of tremor, such as athetosis. These are specially common after the hemiplegia of childhood. The other features are those common to all upper neuron paralyses—viz., (1) the deep reflexes are increased ; (2) there are no electrical changes ; and (3) there is no wasting beyond that of disuse. Finally, *vaso-motor alterations* occur. If the lesion producing the hemiplegia causes irritation of the cortical vaso-motor centres, the hemiplegic limbs are cold, pale, or blue. If, on the other hand, it causes suppression of function, the paralysed limbs are congested, and sometimes œdematous. These considerations

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[1] Rare cases have been recorded in which the paralysis has remained flaccid (hemi-plégie flasque of Bouchard).

Anterior cerebral arteries joined by
anterior communicating artery

Internal carotid
continued onward
as middle cerebral

Lenticulo
striate and
artery of
haemorrhage

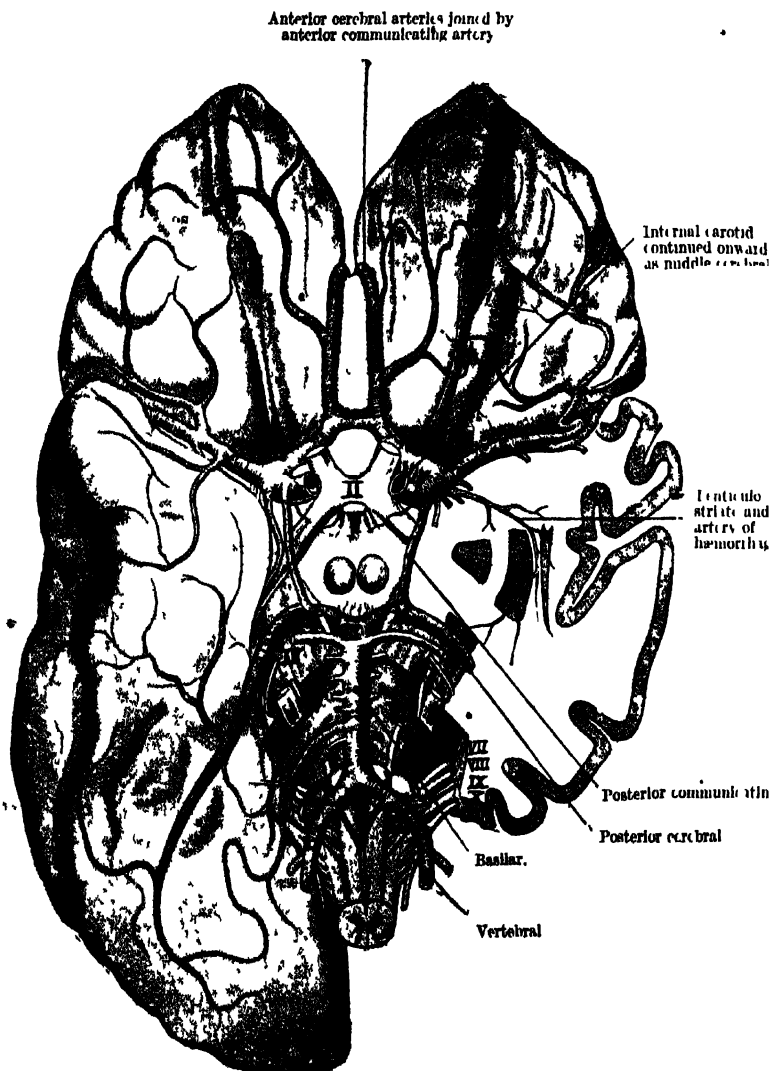Posterior communicating

Posterior cerebral

Basilar.

Vertebral

FIG 159 —THE BASE OF THE BRAIN, showing the arterial distribution and the cranial nerves —
In the oblique section of the left hemisphere are seen from without inwards—grey matter of
the island of Reil, claustrum (grey), external capsule (white); lenticular nucleus (grey),
internal capsule (white) with artery of haemorrhage, and caudate nucleus (grey).   I., Olfactory
lobe; II , optic chiasma ; III , bifurcation of vertebral artery between the third nerves, IV
(on right crus cerebri), beside fourth nerve   V (on pons Varolii), beside fifth nerve ; VI , sixth
nerve (abducens) ; VII , facial nerve ; VIII , auditory nerve ; IX , glossopharyngeal nerve ,
X , vagus or pneumogastric ; XI , spinal accessory ; XII , hypoglossal nerve.

explain the apparently contradictory behaviour of general dropsy, which sometimes avoids and is sometimes most marked in the paralysed limbs ; and the fact that the pulse is sometimes smaller, sometimes larger in the paralysed arm.   The pupil is sometimes contracted on the paralysed side, either from paralysis of the sympathetic or from irritation of the third cranial nerve.   In all organic diseases of the brain which produce hemiplegia there is apt to be some mental disturbance (especially in cortical lesions, such as tumour, hæmorrhage, and meningeal affections).   Aphasia (§ 568) may be associated with right hemiplegia owing to the involvement of the so-called speech " centres " ; in left-handed persor. ·· ft hemiplegia may be so associated.

The *Causes of hemiplegia* may be conveniently divided into those of *sudden* onset—*i.e.*, in the course of a few minutes or a few hours—and those of more *gradual onset*—*i.e.*, in the course of days, weeks, or months.

A *sudden onset* indicates a vascular lesion—

> Hæmorrhage.
> Embolism.
> Thrombosis.
> (In hysterical hemiplegia also the onset is generally sudden.)

A *gradual onset* may be due to—

> Intracranial tumour.
> Abscess.
> Chronic cerebral meningitis and pachymeningitis.
> Thrombosis.

The *age* of the patient is an aid in the diagnosis of the nature of the lesion.

In the *first* half of life suspect in order : Embolism ; thrombosis from syphilitic endarteritis ; hysteria (in females) ; tumour ; abscess.   Cerebral hæmorrhage may occur, even in childhood, but it is rare.

In the *second* half of life in order of frequency we get : Hæmorrhage ; thrombosis from arterial degeneration ; tumour ; aneurysm.

Syphilis plays a very important part in the causation of hemiplegia, which may be produced by (1) a syphilitic endarteritis with thrombosis ; (2) hæmorrhage ; (3) the formation of a gummatous tumour ; (4) meningeal affections.

(a) The **Sudden Causes** (vascular lesions) are much more frequent than those of gradual onset.

(1) INJURY TO THE HEAD may produce hemiplegia by depressed bone, extravasated blood, or, very rarely, by the formation of an abscess or a cicatrix.   It is known by (i.) the history of injury ; (ii.) by being preceded or accompanied by loss of consciousness, stupor, or convulsions ; and (iii.) early rigidity in the affected limbs.

(2) HÆMORRHAGE (apoplexy, § 565) from rupture of a vessel occurs most frequently into the internal capsule (lenticulo-striate artery (Fig. 159, p. 832).   Its differential characters are as follows : (i.) The onset is usually

very sudden, with coma, and often convulsions ; giddiness, or vomiting may mark the advent. (ii.) The patient is usually of middle or advanced life, the blood-pressure is practically always high, and there is frequently a history of chronic interstitial nephritis, though cerebral hæmorrhage is also known to occur in children. (iii) The rigidity of the affected limbs comes on with the hemiplegia, and is succeeded by late rigidity and sometimes by tremors. Meningeal hæmorrhage rarely occurs unless the meninges are previously unhealthy, as in general paralysis of the insane.

(3) EMBOLISM (§ 565), blocking of a cerebral artery, often of the middle cerebral, causes a localised cerebral ischæmia and softening. It is differentiated by (i.) sudden hemiplegia, often accompanied by headache and giddiness ; but (ii.) rarely by convulsions, and as a rule consciousness is not lost ; (iii.) the patient is often young ; (iv.) there is often a history of rheumatic fever, and cardiac valvular disease, especially mitral stenosis ; (v.) the muscles are generally flaccid at first.

(4) ARTERIAL THROMBOSIS (§ 565) consists of coagulation of the blood within an artery owing to its occlusion by disease of the wall. It is favoured by a thrombotic condition of the blood (as in phthisis), and by a sluggish circulation. Senile arterial change, associated with weak heart, is its commonest cause. In younger subjects syphilitic endarteritis is generally the cause. Venous thrombosis is referred to under Coma. Hemiplegia from arterial thrombosis is recognised by (i.) its moderately sudden advent without unconsciousness or convulsions ; (ii.) the paralysed muscles are flaccid at first, *i.e.*, there is no early rigidity ; (iii.) the patient is usually advanced in years, unless the arterial disease be syphilitic. It is held by some that when hemiplegia in old people is survived it is more likely to be due to thrombosis than hæmorrhage.

Arterial Occlusion is the commonest of the cerebral SYPHILITIC LESIONS, and for this reason becomes the commonest cause of hemiplegia under forty. A syphilitic endarteritis gradually occludes an artery, and results in thrombosis and " softening " of a circumscribed area, just as does atheroma of the cerebral vessels. The symptoms of a syphilitic softening resemble atheromatous thrombosis except in two particulars —namely, (1) the extent of the softening, and therefore of the paralysis, if often more imited (*e.g.*, to one arm or one leg) ; and (2) the patient is generally young or middle-aged. A thrombotic hemiplegia in a person under forty or forty-five is almost certainly syphilitic. This kind of cerebral syphilis is distinguished from the meningeal lesions or gumma by the absence of irritative signs, although it is frequently preceded by headache. The advent, contrary to what one would expect from the *gradual* occlusion of an artery, is always more or less sudden, unaccompanied by unconsciousness, convulsions, delirium, rigidity of limbs, optic neuritis, or paralysis of cranial nerves.

(5) HYSTERICAL HEMIPLEGIA is recognised by the following characters : (i.) The advent is usually sudden, and often dates from an emotional shock or hysterical seizure ; (ii.) the paralysis is usually flaccid, incomplete in degree, and the face is exempt ; (iii.) it is often accompanied by hemianæsthesia of the same side, involving general and special sensibility ; (iv.) the condition varies from day to day, and may disappear suddenly, unexpectedly, and completely ; (v.) the youth and sex of the patient are characteristic, and she presents other evidences of the hysterical diathesis (*q.v.*), and a previous history of nervous attacks ; (vi.) the patient does not swing the

leg round in walking; (vii.) in organic hemiplegia when the patient in the supine posture attempts to sit up with folded arms, the extended paralysed leg flexes at the hip joint and the toes of this limb spread out fanwise.

(b) Hemiplegia of **Gradual Onset,** coming on in the course of weeks or perhaps months, points to one of the following conditions : Intracranial tumour, aneurysm, abscess, or more rarely chronic cerebral meningitis (and pachymeningitis). Nevertheless, tumour, aneurysm, and abscess may sometimes give rise to sudden hemiplegia.

§ 589. (6) **Intracranial Tumour,** the sixth cause of hemiplegia, may exist for a long time without any symptoms, especially if the tumour be situated in a silent area. It sometimes gives rise to a progressive and insidious hemiplegia. As regards the nature of the tumour, syphilitic gumma is one of the most frequent in adult life, and is especially apt to affect the meninges at the base or over the convolutions. It is well to bear in mind that malignant tumours may occur in the brain ; sarcoma may occur primarily quite early in life, and a secondary growth of cancer may develop in advancing years. Tuberculous tumours are practically limited to the young. An aneurysmal tumour is not common. Tumours may also arise from the dura.

The *Symptoms* of intracranial tumour vary considerably with its locality (which will be discussed in § 590), but most cases present the following *symptoms in common* : (i.) Headache, vertigo, and vomiting from time to time without nausea. (ii.) Jacksonian epilepsy (§ 635) is frequent if the tumour be situated in or near the motor cortex. (iii.) Œdema of the optic discs (due to increased intracranial pressure), leading to atrophy, is present in a considerable proportion of the cases (five-sixths, Sir William Gowers). The two symptoms—headache and a marked degree of optic œdema, commonly termed optic neuritis—are alone very strongly suggestive of tumour of the brain. (iv.) Paralysis of other cranial nerves, especially of the sixth, fourth, and third, owing to their relatively long course within the cranium. (v.) Incomplete hemiplegia, coming on slowly and insidiously, with rigidity and the other features of upper neuron paralysis (§ 590). Mental apathy and delayed cerebration are usually present.

(7) ABSCESS OF THE BRAIN, the seventh cause of hemiplegia, is rarely primary. It may arise from (1) direct extension, as from disease (syphilitic, tuberculous) of the cranial bones, or after injury or disease of the mastoid, ethmoidal, frontal or nasal sinuses. (2) Abscess also arises from septic emboli, e.g., from malignant endocarditis, or from the lungs when affected with tubercle, bronchiectasis, gangrene, or abscess. It has followed influenza and the specific fevers. The symptoms of cerebral abscess resemble those of tumour, and differ according to the site. Abscess of the temporal lobe after middle-ear disease is not uncommon, and in this case the hemiplegia is of slow onset, ill-defined, and the face is notably involved. The other symptoms are described in Intracranial Inflammation, § 585. Pyrexial symptoms are only found at the onset and the termination of the case.

(8) CHRONIC MENINGITIS AND PACHYMENINGITIS (fibrous thickening of the

meninges) sometimes occur (though rarely) as solitary lesions. The condition is generally due either to chronic alcoholism, syphilis, or injury, and some say that in the absence of these, chronic meningitis may be excluded. Tuberculous meningitis may also cause hemiplegia, especially in children. ʹ (i.) The advent is usually gradual; (ii.) the degree of the hemiplegia is slight ; (iii.) optic neuritis is frequently present ; (iv.) very generally some of the cranial nerves are involved as they pass through the membranes. (v.) There are always some signs of mental alteration, and not infrequently epileptiform convulsions from irritation of the cortex. Epilepsy occurring for the first time in a person of thirty or forty is very probably due to syphilitic disease of the meninges.

Syphilitic Meningeal Affections may also take the form of a gummatous deposit. These are distinguished clinically by severe and continuous headache, and the presence of irritative signs.

(9) ALTERED BLOOD STATES, such as those due to chorea, severe anæmia, pregnancy, typhoid fever, influenza, scarlet and other fevers, may give rise to hemiplegia, probably thrombotic in nature.

(10) Hemiplegia may also occur in disseminated sclerosis and general paralysis of the insane.

Prognosis of Hemiplegia.—If the paralysis has been considerable, one can hardly hope for complete recovery. The most favourable kinds, both as regards recovery and life, are those due to injury and hysteria. All the other causes are more serious. Localised cerebral tumours are sometimes removable, and life may be prolonged, but the paralysis rarely disappears entirely. Abscess is capable of surgical treatment. The most unfavourable kinds of hemiplegia are those due to embolism, thrombosis, and hæmorrhage, the last-named being as regards life the gravest of all ; and the usual history in these cases, even if the patient recover from the apoplexy, is that a recurrence takes place during the ensuing year or two, in which the patient dies. Some contend that cerebral hæmorrhage is rarely recovered from. As regards symptoms, it is usual for the leg to recover before the arm ; if the converse of this happens, the prognosis is less favourable. The formation of blisters or sloughs over the gluteus is of bad omen.ʹ The general condition of the patient, and the existence of some other disease, such as chronic granular kidney, may make the outlook bad.

Treatment of Hemiplegia.—In hæmorrhage the patient should be kept at perfect rest at first ; the treatment of hæmorrhage, embolism, and thrombosis should be directed to the cardio-vascular system (see § 565). Active steps should be taken to reduce a high blood-pressure. Intracranial tumour and abscess, unless we can positively exclude syphilis, should be put on full doses of iodide and mercury while the locality is being accurately diagnosed, with a view to surgical interference should these remedies fail. Headache and the other symptoms of tumour may be relieved by lumbar puncture (p. 952, § 666), and by decompression. In all cases of severe headache or optic neuritis from cerebral tumour decompression should be resorted to ; in the latter case in order to save the sight. For aneurysm full doses of iodide of potassium are useful. Chronic meningitis, if of a syphilitic nature, should be treated accordingly ; if due to injury, and

the position of the focus of the disease can be diagnosed, surgery may be invoked. As regards the treatment of the paralysed limbs, a great deal can be done by the judicious application of massage, galvanism, and galvano-faradism. The first improves the nutrition of the muscles considerably, the second and third maintain and improve their functional activity. Galvanism only may be applied to rigid muscles, and general hygienic measures will aid. Electricity should not be started till two months after a cerebral hæmorrhage, and should be stopped if headache, faintness, or fatigue appears. If the motor tract is definitely destroyed, nothing will restore its functions completely, but a good deal may be done by the means just mentioned, and by the education of other centres to take on the functions of those destroyed. Last, but not least, shortening of the muscles in the paralysed limbs should at an early stage be prevented by means of splints.

§ 590. The **Localisation of Intracranial Lesions** may be considered in regard to : firstly, certain recognised types of paralysis ; secondly, certain groups of symptoms corresponding to the three basal fossæ ; and, thirdly, the symptoms pointing to positions in the encephalon.

A. Lesions in certain positions give rise to certain TYPES OF PARALYSIS.

(1) Hemiplegia is usually due to a lesion in the internal capsule.

(2) " Crossed hemiplegia " (face on same side as lesion, body on opposite side) is typical of a lesion in the lower part of the pons after the facial tract has crossed.

(3) Paralysis of the facial and sixth nerves on same side as the lesion, hemiplegia of opposite side, signifies lesion in pons at level of exit of facial nerve (Millard-Gübler syndrome).

(4) Incomplete paralysis of the third nerve on same side, with hemiplegia or hemiataxy on the opposite side, is typical of a lesion in the crus cerebri (Weber's syndrome).

(5) Oculo-motor paralysis of the same side as the lesion, with tremor of the leg and arm on the opposite side, indicates a superficial lesion on one crus extending into the tegmentum and the neighbourhood of the red nucleus (Benedikt's syndrome).

(6) Conjugate deviation of the eyes indicates a lesion at or, more generally, above the ocular nuclei. With destructive lesion of the cortex the eyes " look towards the lesion," but they look *away* from *irritative* lesions.

B. Intracranial tumours in the—

CORTEX of the brain (see Figs. 145 and 146, § 533). Tumour in or near the pre-central gyrus causes " Jacksonian epilepsy." The convulsions begin in the part controlled by the affected area, the hand, face, or foot being the most frequent starting-points. If the convulsions spread to the opposite side there is generally loss of consciousness. Immediately after such an attack the side opposite the lesion is often left temporarily hemiplegic and anæsthetic, especially in the hands and feet. When Jacksonian epilepsy begins in the right side of the face or right hand it may leave temporary aphasia. Destructive lesions of the pre-central gyrus may cause monoplegia, or hemiplegia. Destructive lesions of the speech centres may give rise to aphasia (motor or sensory). Tumours of the special sense centres may give rise to special sense aurae, e.g., flashes of light, noises, odours.

POSTERIOR FOSSA—resemble symptoms of cerebellar (below), pontine, and medullary tumours, but without conjugate deviation. Cranial nerves from VI. to XII. may be involved (Fig. 159, p. 832).

MIDDLE FOSSA—lesion of one third nerve, and symptoms of involvement of crus cerebri (hemiparesis).

ANTERIOR FOSSA—paralysis of nerves entering the orbit, loss of sight and smell on side of lesion. Sometimes no symptoms except mental d⬛⬛⬛.

C. Destructive lesions in different positions in the encephalon are indicated as follows—

FRONTAL LOBE—mental torpor and depression, sometimes exophthalmos on the side of a tumour, and perversion of smell. Usually no motor or sensory disturbance.

OCCIPITAL LOBE—altered field of vision (hemianopia); if bilateral, total blindness, but blindness from this cause is almost unknown. Left angular gyrus, word-blindness.

TEMPORO-SPHENOIDAL LOBE—in left superior convolution, word-deafness; if tip, taste and smell affected.

CORPUS CALLOSUM—(i.) Gradual onset of hemiplegia, with vague hemiplegic symptoms on other side; (ii.) mental torpor, apraxia, coma and death without involvement of cranial nerves; (iv.) headache and vomiting are rare.

INTERNAL CAPSULE—hemiplegia, face and body, of opposite side. When posterior part is involved, hemianæsthesia, hemianopia, disturbed hearing, tremor (especially when optic thalamus also involved), hemichorea, and athetosis. In a linear (antero-posterior) lesion, paralysis of face and leg, arm escaping.

OPTIC THALAMUS—hemianopia, hemiataxi and hemianæsthesia. Persistent paroxysmal pain on affected side. There is an exaggeration of the emotional content of both pleasant and unpleasant sensations. Thus slight heat applied to the hemiplegic side would be felt as intense burning. Little or no hemiplegia. Irregular athetoid or choreic movements of affected limbs. These symptoms constitute th. *thalamic syndrome of Déjerine.*

CORPORA QUADRIGEMINA—*superior* corpora, loss of pupillary contraction to light and accommodation, ataxy if fibres to medial lemniscus involved. *Inferior corpora*: auditory disturbance, partial deafness of both and especially of opposite ear. Defective mastication from involvement of motor root of V., and the fourth nerve is sometimes involved. Lesions of corpora quadrigemina simulate locomotor ataxy or cerebellar tumour. Ophthalmoplegia and reeling gait, especially if associated with bilaterally defective hearing, render diagnosis probable.

CRUS CEREBRI—simultaneous onset of III. nerve paralysis on side of lesion, and of hemiplegia (with marked facial involvement) on opposite side, is very characteristic ; and if *tegmen* involved, hemiataxy of opposite side.

PONS—very varied symptoms ; most characteristic are combination of paralysis of V., VI., and VII. on side of lesion, and of body on opposite side. *Upper part of pons* : Paralysis and anæsthesia of face and body on side opposite to lesion. *Lower part of pons* : " crossed paralysis," face on side of lesion, body on opposite side ; VI. and VII. paralysed together ; conjugate deviation to side away from destructive lesion ; in acute lesions, contracted pupils and hyperpyrexia.

MEDULLA—difficulty in articulation and swallowing, associated with disturbances of heart and respiration and paresis or paralysis of limbs on one or both sides. All cranial nerves from VIII. to XII. affected. Often secondary to cerebellar or pontine disease.

CEREBELLUM—ataxy (reeling), static and dynamic, not affected by closing eyes ; movements fairly co-ordinated when in bed. Vertigo. Nystagmus. Paresis of trunk or limbs. Hydrocephalus by pressure on veins of Galen leading to distended ventricles. Compare § 617.

PITUITARY BODY—Bilateral hemianopsia ; acromegaly or hypopituitarism.

§ 591. **Hemiplegia in Children** differs from that in adults in several respects. It is met with in congenital and in acquired forms, the latter occurring usually under ten years of age—the child has a sudden fit of convulsions, followed by coma and hemiplegia. In after life, athetosis and post-hemiplegic chorea are common sequelæ, and Jacksonian epilepsy is also seen. The affected limbs do not grow so well as the others. The mental condition in such patients varies—it may be normal or the child may be merely dull. More rarely idiocy results, often with squint.

The *Prognosis* as to recovery is bad, but the duration of life is not affected by this

disease except in the congenital and foetal cases, who frequently die in early life from intercurrent disease.

Etiology.—Congenital hemiplegia results from injury at birth, causing meningeal haemorrhage or bruising of the hemispheres (Little's paralysis) ; it may be associated with porencephalus (cavities in the brain) and atrophy of one hemisphere. Acquired hemiplegia in children comes on in the majority of cases before the second year. The hemiplegia in such cases is apt to develop during or after an acute specific fever, and has been found after death due to embolism, thrombosis, or haemorrhage. Tuberculous meningitis and tumour, cerebral abscess, polio-encephalitis superior and trauma are also causes of hemiplegia in children.

Treatment.—The possibility that infantile convulsions are associated with a cerebral lesion should be remembered, and bromides and a purge should be given. The hemiplegia may profitably be treated by electricity and massage. The healthy upper limb should at times be kept in a splint, so that the child may be compelled to exercise the affected one. The rigidity and contractures are treated by massage and exercises. And see Treatment, p. 836.

The patient complains of weakness or **paralysis** *of* BOTH LEGS. The disease (general debility being excluded) is PARAPLEGIA.

§ 592. **Paraplegia** is most often due to structural disease of the spinal cord, for it is here that the motor tracts of the two sides of the body run side by side, and can therefore be affected by a single transverse lesion. Paraplegia may also be due to peripheral neuritis.

It is convenient for clinical purposes to make three groups of paraplegias :

(a) The **upper neuron, spastic or rigid paraplegias.** The paralysis is persistent, the muscles are rigid, with no tendency to atrophy, beyond that due to disuse, the deep reflexes are increased, Babinski's sign and ankle clonus are present, and there are no electrical changes .. § 593

(b) Paraplegias of the **lower neuron** type are **flaccid.** The paralysis is persistent, the muscles are atrophied, the deep reflexes are absent or diminished, and there is the reaction of degeneration .. .. § 597

(c) The **functional** or **variable paraplegias.** The paralysis is less pronounced than in the foregoing ; it is apt to vary from day to day, and neither atrophy nor electrical changes are present .. .. § 600

CLINICAL INVESTIGATION.—Assuming that the case is really one of paraplegia, and not simply a stiffness of the joints due to gout, rheumatism, or old age, it is desirable (1) to investigate the history of the case, (2) to examine the spinal column, and (3) to ascertain whether the paralysis conforms to the upper neuron type, the lower neuron type, or the functional type.

(1) The history of the case—particularly in regard to the mode of onset and evolution, the history of pain, and the age of the patient—throws considerable light on the nature of the lesion. The onset is rapid in vascular lesions or those of an acute inflammatory type. It is more gradual in some forms of compression paraplegia (such as Pott's disease (§ 593)), multiple neuritis, chronic myelitis, and syringomyelia. The history of pain in the spine or legs is of importance, and some even go so far as to divide paraplegias into painful and painless. The presence of pain indicates an involvement of the nerve trunks or the posterior nerve roots, as in meningeal affections and tumours. Paraplegia in *childhood* in a large proportion of cases is due to Pott's disease or infantile palsy. *Adults* from twenty to forty are chiefly affected by paraplegia due to spinal syphilis, multiple neuritis, or hysteria. In *advanced* life the slow degenerative lesions are more often met with.

(2) Local examination of the spine should never be omitted. It will show us at a glance whether or not angular curvature or malformation be present. Percussion down the spine may elicit tenderness, as in Pott's disease, tumour or meningeal affections. Vertical pressure upon the head while the patient is sitting or standing will cause severe pain if vertebral caries or cancer be present, but not in hysterical or neurasthenic conditions. The spine is kept rigid in all painful organic affections of the spine, as in those of the spinal meninges. X-ray examination may be helpful.

(a) The **upper neuron, spastic,** or **rigid paraplegias** form the largest of the three groups. The three features of this kind of paraplegia are those which belong to all paralyses due to lesions of the upper neuron—viz., rigidity, increased deep reflexes, and absence of decided muscular wasting. The members of this group differ from the functional paraplegias in their steady and usually progressive course. The following are the causes of the spastic paraplegias, placed somewhat in order of frequency :

Compression Paraplegias.

    I. Pott's disease.
   II. Tumours.
  III. Injury.

Inflammation of the Cord or its Membranes.

  IV. Myelitis and its varieties.
   V. Hæmorrhage (spinal and meningeal).
  VI. Embolism.
 VII. Chronic spinal pachymeningitis.

Chronic System Lesions (Sclerosis).

VIII. Disseminated sclerosis, general paralysis of the insane, and occasionally tabes dorsalis.
  IX. Primary lateral sclerosis.
   X. Ataxic paraplegia and Friedreich's disease (occasionally).
  XI. Amyotrophic lateral sclerosis.
 XII. Infantile cerebral and spinal paraplegia and other double cerebral or cerebellar lesions.
XIII. Toxic sclerosis.

Malformations.

***

§ 593. Compression Paraplegia is one of the commonest forms of **spastic paraplegia,** and is due to compression of the spinal cord, either by spinal caries (Pott's disease), or other disease or injury of the vertebra, or a tumour pressing upon the spinal cord.

The *Symptoms* common to all forms of compression paraplegia (in addition to those just mentioned as belonging to upper neuron paraplegias), are (1) pain from pressure upon the nerve roots, and, generally speaking, it may be said that when the pain precedes the paraplegia the lesion is extradural ; when the converse, that the lesion is probably intradural. 2) The symptoms are apt to be more marked in one leg than in the other (with the notable exception of Pott's disease).

FUNCTIONS OF THE SPINAL SEGMENTS.

Segment of Cord and Spinous Process	Muscles Supplied	Reflex.	Sensation.
II and III C. Opposite 1st C	Sterno-mastoid. Trapezius. Scaleni and neck. Diaphragm.	Sudden inspiration produced by sudden pressure beneath the lower border of ribs.	Back of head to vertex. Neck.
IV C Opposite 2nd C	Diaphragm. Deltoid. Biceps. Coraco-brachialis. Supinator longus. Rhomboids Supra- and Infra-spinatus Levator ang. scapulæ.	Pupil reflex — 4th to 7th cervical—i.e, dilatation of the pupil produced by irritation of neck.	Neck Shoulder. Outer arm.
V C Opposite 3rd C.	Deltoid. Biceps Coraco-brachialis Brachialis anticus. Supinator longus and brevis Rhomboids and teres minor. Pectoralis (clavicular part). Serratus magnus.	Scapular reflex 5th cervical to 1st thoracic -i e., irritation of the skin over the scapula produces contraction of the scapular muscles. Tapping tendon of supinator longus in wrist produces flexion of forearm.	Back of shoulder and arm. Outer side of arm and forearm, front and back.
VI. C Opposite 4th C.	Biceps Brachialis anticus. Pectoralis (clavicular part). Serratus magnus. Triceps. Extensors of wrist and fingers. Pronators.	Triceps reflex — 5th to 6th cervical—i.e., tapping elbow tendon produces extension of forearm. Posterior wrist reflex — 6th to 8th cervical —i.e , tapping tendons causes extension of hand.	Outer side of forearm, front and back. Outer half of hand.
VII C Opposite 5th C	Triceps (long head). Extensors of wrist and fingers. Pronators of wrist. Flexors of wrist. Subscapularis. Pectoralis (costal part). Latissimus dorsi. Teres major.	Anterior wrist reflex 7th to 8th cervical —i.e., tapping anterior tendons causes flexion of wrist. Palmar — 7th cervical to 1st thoracic—i.e , stroking palm causes closure of fingers.	Inner side and back of arm and forearm Radial half of the hand.
VIII. C Opposite 6th C.	Flexors of wrist and fingers. Intrinsic muscles of hand.		Forearm and hand, inner half.
I D. Opposite 7th C.	Extensors of thumb. Intrinsic hand muscles. Thenar and hypothenar eminences.		Forearm, inner half Ulnar distribution to hand.
II. to XII. D. Opposite 1st to 10th D.	Muscles of back and abdomen. Erectores spinæ.	Epigastric reflex = 4th to 7th thoracic—i e , irritation of mammary region causes retraction of epigastrium. Abdominal = 7th to 11th thoracic—i.e , stroking side of abdomen causes retraction of belly.	Skin of chest and abdomen in bands running around and downward, corresponding to spinal nerves. Upper gluteal region.

FUNCTIONS OF THE SPINAL SEGMENTS—*continued.*

Segment of Cord and Spinous Process.	Muscles Supplied.	Reflex.	Sensation.
I. L. Opposite 11th D.	Ilio-psoas. Sartorius. Muscles of abdomen.	Cremasteric = 1st to 3rd lumbar—*i.e.*, stroking inner thigh causes retraction of scrotum.	Skin over groin and front of scrotum.
II. L. Opposite 11th D.	Ilio-psoas. Sartorius. Flexors of knee (Remak). Quadriceps femoris.	Tapping patellar tendon causes extension of leg.	Outer side of thigh.
III. L. Opposite 12th D.	Quadriceps femoris. Inner rotators of thigh. Abductors of thigh.		Front and inner side of thigh.
IV. L. Opposite 12th D.	Abductors of thigh. Adductors of thigh. Flexors of knee (Ferrier) Tibialis anticus.	Gluteal ·· 4th to 5th lumbar—*i.e.*, stroking buttock causes dimpling in fold of buttock.	Inner side of thigh and leg to ankle. Inner side of foot.
V. L. Opposite 12th D.	Outward rotators of thigh. Flexors of knee (Ferrier). Flexors of ankle. Extensors of toes.		Back of thigh, back of leg, and other part of foot.
I. to II. S. Opposite 1st L.	Flexors of ankle, and toes. Peronei.	Plantar reflex.	Back of thigh. Leg and foot, outer side.
III. to V. S. Opposite 1st L.	Perineal muscles.	Ankle-clonus. Bladder and rectal centres.	Skin over sacrum. Anus. Perinæum. Genitals.

I. **Vertebral Caries** (Pott's Disease, Tuberculosis of the Spine) may be regarded as the type of compression-paraplegias. It is the commonest of these paraplegias, injury coming next, and vertebral cancer next ; aneurysm and exostosis are rare causes. The paraplegia in Pott's disease is due to the pressure on the cord of the inflammatory products which accumulate outside the dura mater. Its differential features are : (1) In the incipient stage the patient, usually a child, probably complains of " stomachache " or pains in the region of the umbilicus, the back, or the loins, due to pressure on the nerve roots. A sense of constriction round the trunk may be experienced. (2) The onset of the paralysis is usually gradual, and is rarely quite complete, but on rare occasions it is rapid. Both legs are affected equally. (3) There are marked rigidity and increase of the deep reflexes, and often twitchings of the muscles ; sensation is generally retained. (4) In the early phases, the jarring produced by percussion on the head or by jumping on the heels causes pain ; pain and tenderness of the spine are generally present. (5) The subjects of the disease are mostly children or young persons who have a family or antecedent history of tuberculosis, and perhaps other manifestations of that disease. (6) The course is always protracted. Spinal caries, even without operation, tends

in the long run to become quiescent, and if the patient is able to undergo
prolonged rest, it is wonderful how the use of the legs may be restored,
even after complete paralysis.• I have seen cases recover after a complete
paralysis lasting two or three years, and have made an autopsy on a.
patient whose cord at one spot was no thicker than a quill.   The *Diagnosis*
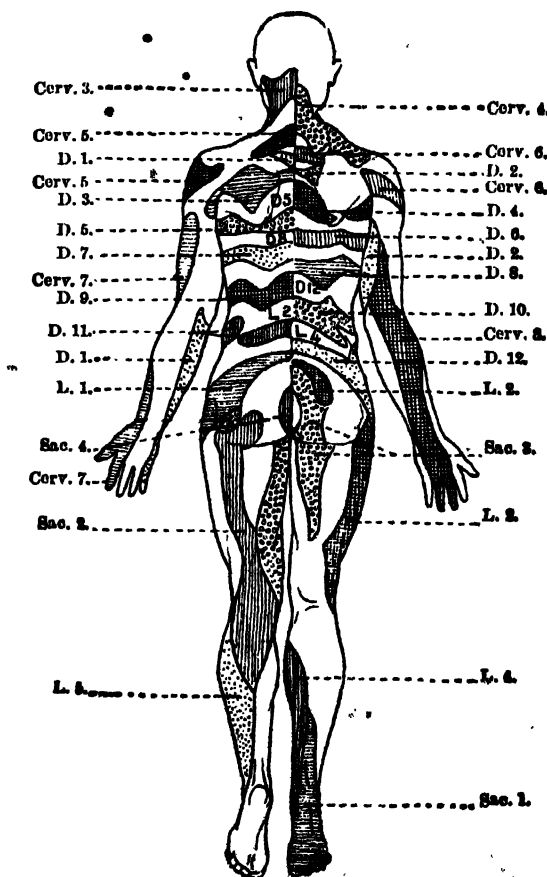is only difficult when there is no curvature or other local indication of

FIG. 160.—SEGMENTAL ALGESIC AREAS OF HENRY HEAD—*i.e.*, areas of increased or diminished
cutaneous sensibility to pain when the corresponding spinal segment is irritated or destroyed
respectively.

caries and then can be cleared up by an X-ray examination.   Paraplegia
from cancer occurs in older subjects.   Exostoses can only be suspected
when found in other parts of the body.   These and all the other causes
of compression-paraplegia are distinguished from Pott's paraplegia by
(1) the unilateral predominance of the symptoms, and (2) pain being
relatively a more prominent symptom.   Von Pirquet's and other reactions

for tuberculosis may be of assistance (§ 110). For the diagnosis of the position of the lesion, see below. •

II. **Compression-Paraplegia due to Spinal Tumour.**—Tumours of the spinal cord may be (a) extra-medullary (arising outside the spinal cord) or (β) intra-medullary (arising within the spinal cord). Extra-medullary tumours are the more common, and it is these which produce typical compression-paraplegia.
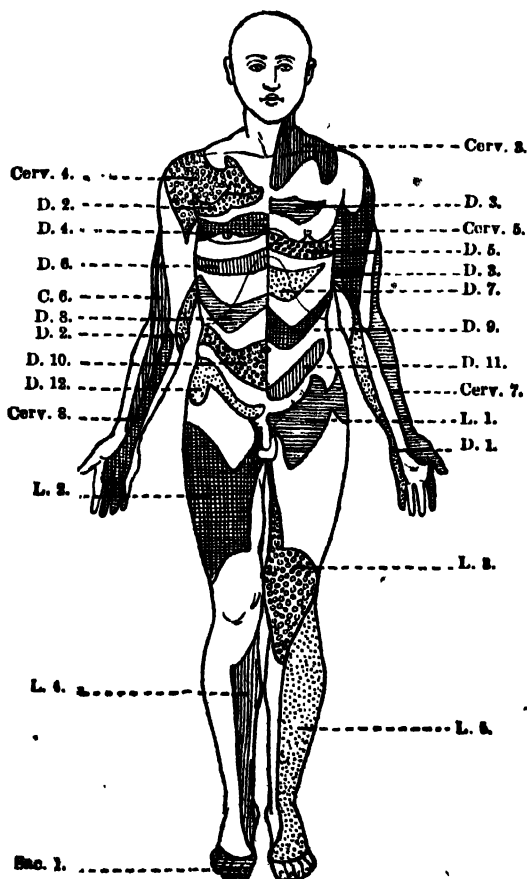

FIG. 161.—Front view of Fig. 160.

(a) EXTRA-MEDULLARY SPINAL TUMOURS may be extra- or intra-thecal, but these wo cannot be clinically distinguished. The following are the principal extra-medullary tumours—gumma, sarcoma, carcinoma, tubercular growths, myxoma, fibroma, hydatid cysts, chondroma, exostoses, lipoma (rare), neurofibroma (springing from the nerve roots in the canal). Aneurysm, carcinoma, and sarcoma may arise outside the vertical column, and invade the cord after eroding the vertebræ, but these three lesions generally have their own proper symptoms.

Symptoms of Extra-medullary Spinal Tumours.—(1) Pain radiating in the distribution of the nerve roots pressed on is usually the earliest indication, and is rarely

absent. Generally it is one-sided, round the chest or abdomen, or down the leg; sometimes there is pain at the site of the tumour. (2) Progressive paraplegia *starting and predominating in one leg*, and spreading upwards from the toes, more marked on the same side as the pain, and associated with (3) anæsthesia having the same features. Finally, both the paralysis and the anæsthesia become complete up to a definite level. The upper limit of the anæsthesia is the best guide as to the seat of the tumour, bearing in mind that the posterior roots enter the cord two or more inches (in the dorsal region) above their entrance into the spinal canal (Fig. 160), and therefore the tumour must be cut down upon well above the anæsthesia limit. Of the two sub-groups of extra-medullary tumours, *extra-thecal lesions* are more often attended by pain, and *intra-thecal lesions* by cramps and contractures of the muscles at night, indicating compression myelitis. The superficial abdominal reflexes are sometimes abolished on the same side as the tumour—the supra-umbilical reflexes in tumours between the eighth and ninth, the infra-umbilical in tumours lying between the eleventh and twelfth dorsal vertebræ. Sometimes the umbilicus is drawn towards the healthy side. In meningeal hæmorrhage the symptoms of pain and paralysis are of sudden onset.

The *Diagnosis* of such cases rests on the presence and character of the pain, the progressive evolution of the symptoms, and their predominance on one side. Cervical meningitis causes pain on both sides. The *Prognosis* depends mainly on the nature and position of the tumour, and whether secondary myelitis has occurred. The *Treatment* (apart from anti-syphilitic remedies) is surgical.

β. SYRINGOMYELIA AND INTRA-MEDULLARY TUMOURS (§ 644) sometimes come under notice for paralysis of the legs, and in this paraplegia there may be the peculiar association of an *atrophic paralysis with rigidity and increased deep reflexes*, owing to the mutual involvement of the anterior horns and lateral columns, which may cause atrophy of some muscle-fibres (*e.g.*, of the vasti) and rigidity of others. The earliest symptoms of syringomyelia usually appear in the arms (sensory and trophic changes). The symptoms of an intramedullary tumour necessarily vary according to the position of the tumour and the column chiefly involved.

III. **Injury to the Spinal Column** may cause displacement of or damage to the vertebræ, or hæmorrhage, or it may initiate acute myelitis or meningitis (*q.v.*). The symptoms following injury are those either of destruction (paralysis), or irritation (spasm).

In cases of transverse myelitis after fracture-dislocation, it was formerly taught that the reflexes below the lesion are exaggerated, but Charlton Bastian has shown that in complete functional severance of the cord, whether from actual division or transverse myelitis, they may be absent; they may, however, return, as in a case recently recorded by F. Buzzard.

§ 594. The **Localisation of Spinal Lesions**, (*a*) as regards its LEVEL, is ascertained by observing: (1) The distribution of the motor weakness; (2) the alteration of the reflexes; and (3) the level of the upper limit of the pain and anæsthesia (see Head's Areas, below). For (1) and (2) consult the table on pp. 841, 842.

(*b*) The particular COLUMN which is affected can be ascertained by studying the table in § 532.

Head's Areas of Analgesia and Hyperalgesia (pp. 843 and 844) may be of some use for the localisation of a spinal injury or lesion, but they are difficult to elicit and define. They have been determined by the successive researches of James Ross,[1] James Mackenzie,[2] William Thorburn,[3] and, more particularly, Henry Head,[4] whose

[1] *Brain*, January, 1888, part x., p. 333; and "Diseases of the Nervous System."
[2] *Brain*, 1893, part xvi., pp. 321 and 515; and *Med. Chronicle*, August, 1892.
[3] *Brain*, 1893, part xvi., p. 355.
[4] *Brain*, 1893, part xvi., p. 1; 1894, part xvii., p. 339; 1896, part xix., p. 153.

conclusions are founded upon an investigation of (1) cases of injury to the spinal nerve roots, (2) cases of visceral disease associated with soreness of the skin or reflex pain, and (3) cases of herpes. They consist of areas of diminished or increased *sensibility to pain*, corresponding to certain spinal segments, as indicated in Figs. 160 and 161. The reader is asked to remember in what follows that we are not concerned with tactile sensation, but only with sensibility to pain (algesia). A destructive lesion of a spinal segment or nerve root is attended by analgesia of the corresponding area, an irritative lesion by hyperalgesia. To localise a spinal lesion you should carefully test the sensibility from above downwards with the point of a pin, and you will find an *abrupt margin of normal sensibility to pain* at the upper border of the affected area. The important question is to what level does the sensibility to pain remain normal.

These areas may also have two other clinical uses. In certain *visceral diseases* two or more of them are apt to be the *seat of tenderness*, and even pain, which is probably of a reflex nature ; single area tenderness is practically never met with. To test these areas the rounded head of a hat-pin is convenient, its smooth surface evoking the sensation of soreness. The various organs stand in connection mainly with the areas as shown in table below, the areas being those indicated in the figures (Dr. Henry Head). There are also certain smaller *spots* of maximum tenderness to which a patient chiefly refers his pain ; these remain tender longer than the rest of the skin around. The whole of this question is at the present time one more of scientific than practical interest.

TABLE OF VISCERAL DISEASE IN RELATION TO SUPERFICIAL TENDERNESS OR REFLECTED PAIN (DR. HENRY HEAD).

Heart, Ventricle .	Dorsal 1 (?), Dorsal 2, 3, 4, 5.
Auricle . .	Dorsal 5, 6, 7, 8, and (?) 9.
Aortic Arch . 4.	Cervical 3 and 4, Dorsal 1, 2, 3, 4.
Dorsal Aorta .	Dorsal 5, 6, 7, 8, 9.
Abdominal Aorta .	Dorsal 10, 11, 12, and Lumbar 1.
Lungs . . .	Cervical 3, 4, Dorsal 3, 4, 5, 6, 7, 8, 9.
Œsophagus . .	Dorsal 5, 6, 7.
Stomach . .	Dorsal 6, 7, 8, 9, 10.
Intestine—	
1. Duodenum to Sigmoid Fl.	Dorsal 10, 11, 12.
2. Rectum	Sacral 2, 3, 4.
Liver and Gall Bladder	Dorsal 7, 8, 9, 10. (Right side.)
Kidney and Ureter	Dorsal 10, 11, 12, Lumbar 1, Lumbar 2 and Occipital.
Bladder	Sacral 2, 3, 4.
Prostate	Sacral 2, 3, 4, Dorsal 10 and (?) 11 and Occipital.
Testicle	Dorsal 10.
Epididymis	Dorsal 11 and 12.
Ovary .	Dorsal 10.
Uterine Appendages	Dorsal 10, 11, 12, Lumbar 1, and (?) Lumbar 2.
Cervix Uteri and Lower Segment of Uterus	Sacral, 2, 3, 4.

Area on the Scalp.	*Area on th Trunk.*	*Organs in relation with these Areas.*
Fronto-nasal.	Cervical 3 Cervical 4	} Lung (Apices), Stomach, Liver (Occipital).
Mid-orbital.	Dorsal 2. Dorsal 3. Dorsal 4.	} Heart (Ventricle), Aortic Arch, Lungs.
Fronto-temporal.	Dorsal 5.	Lung, Heart.
	Dorsal 6.	Lungs (lower lobes), Heart (Auricle).
Temporal.	Dorsal 7.	Lungs (lower lobes), Heart (Auricle), Stomach (Cardiac end), Liver (R. side).
Vertical.	Dorsal 8.	Stomach, Liver, and Gall Bladder, Lungs.
Parietal.	Dorsal 9.	Stomach (Pyloric end), Liver.
Occipital.	Dorsal 10.	Intestine, Liver, Ovary, Testis, Stomach (Occipital).

● Head's areas also correspond to the areas affected with *herpes* (zoster), which is now proved to be due to some irritative lesion of the posterior root-ganglion. It was, indeed, the careful study of a large number of cases of herpes and the examination

of twenty-one fatal cases which enabled Head to map out these algesic areas with precision.

§ 595. IV. **Acute Transverse Myelitis** is a transverse softening of the cord, due to inflammation or thrombosis, characterised in typical cases by complete loss of sensation and motion in the parts supplied by-the cord below the upper level of the lesion, by the " girdle " pain, and by the tendency to bedsores and sphincter troubles. The dorsal region is the most usual position of the lesion. This paraplegia *at first* is flaccid.

Symptoms.—1. The advent may be as sudden as apoplexy, or it may occupy a few days. There is generally slight pyrexia. According to the severity of the onset, cases are described as acute or subacute. 2. The " girdle " pain is a characteristic symptom, consisting of a feeling as of a constricting cord around the trunk corresponding to the upper limit of the lesion. There is also a band of hyperæsthesia and increased superficial reflexes in that position. 3. Below this level sensation and motor power are lost. Atrophy of the muscles supplied by the affected region of the cord supervenes, *e.g.*, of the upper limbs when the cervical enlargement is affected and of the lower limbs when the lumbar enlargement is affected (§ 637). The paralysis is flaccid in the early stage, but the muscles supplied by the cord below the lesion gradually become stiff in the course of a few weeks owing to the descending sclerosis. 4. The descending sclerosis leads to increase of the deep reflexes below the lesion. The reflexes, superficial and deep, are lost in the parts supplied by the affected region of the cord. 5. Retention of urine and fæces is present at the onset ; later on evacuation takes place without the patient being conscious of it. If the lumbar centres are involved, there is true incontinence from the onset. Bedsores nearly always supervene, *unless carefully guarded against from the beginning*, especially in cases where the lumbar region is involved. 6. The course is always rapid, and there is but little hope of regaining full use of the affected limbs. In an ordinary case sensation may return in three to six months, and motion to some extent in six to eighteen months. The higher up or lower down the lesion, the more grave the prospect. When the cervical region is affected, death often occurs in a few days from pneumonia, or paralysis of the respiratory muscles. When the lumbar enlargement is affected, death may ensue from cystitis, surgical kidney, or bedsores.

The *Diagnosis* is not, as a rule, difficult, on account of the girdle pain, the rapid involvement of the bladder and rectum, and the completeness of the paralysis and anæsthesia. The pyrexial onset, with headache, etc., aids in the diagnosis from hæmorrhage and embolism (see below).

Causes.—Acute myelitis is most common in males between ten and forty years of age. It is now held that most cases of so-called " transverse myelitis " are really cases of softening (myelomalacia) from thrombosis (*e.g.*, from syphilitic endarteritis). Among other causes may be mentioned injury, hæmorrhage, extension of inflammation from the meninges, exposure, and various toxic blood states.

Chronic Transverse Myelitis may supervene on the acute, or it may be chronic from the onset. It presents the same characteristics as acute myelitis in a lesser degree. At first, before the rigidity supervenes, it may resemble multiple neuritis.

Acute diffuse or Central Myelitis is a rare form of acute myelitis, accompanied by marked pyrexia, widespread paresis, and anæsthesia (§ 609). It spreads rapidly to the whole·cord, and is generally fatal.

§ 596. Vascular and other Lesions of the Spinal Cord.—V. **Hæmorrhage** into the spinal cord is said by most observers to be very rare, unless preceded by some congenital cystic defect or some degenerative or neoplastic lesion, such as glioma. The exacerbations of symptoms in syringomyelia are believed to be due to hæmorrhage. Intra-medullary hæmorrhage is characterised by the sudden onset of complete motor and sensory paralysis. It presen˚ ˙st of the symptoms of acute myelitis, from which it can only be differentiated by its *instantaneous occurrence, attended sometimes by severe localised pain*. Sometimes there is a history of trauma. Hæmorrhage into the spinal membranes causes similar symptoms, accompanied by pain due to pressure on the nerve-roots.

VI. **Embolism of the Cord** is rare. The patient complains of a severe " shock in the spine," followed by sudden and complete paralysis within a definite area, corresponding to the position of the lesion. Cardiac disease or other causes of embolism are in operation.

VII. **Spinal Pachymeningitis** (Chronic Spinal Meningitis, Meningeal Thickening) may give rise to paraplegia, with stiffness of the legs, of very gradual onset and prolonged course. This morbid condition is said by some to be infrequent, and not to give rise to any obvious symptoms, but at the Paddington Infirmary I had no difficulty in collecting seven cases (verified by autopsy) in the course of two or three years.

1. The leading *Symptoms* of this form of paraplegia is pain shooting down the nerves of the leg and elsewhere, aggravated by any movement of the back ; sometimes extremely severe, and accompanied by tenderness of the spine. 2. Stiffness and weakness of the legs, involuntary twitchings and increased reflexes were present in my cases as the disease progressed. 3. Patches of hyperæsthesia and anæsthesia were occasionally present, and in some there was a progressive atrophic weakness of various muscles due to the constriction of the nerve roots. 4. In certain cases the thecal mischief spreads to the spinal cord, and various symptoms may arise according to the column involved. When the posterior column is affected, as in the cases alluded to in § 615, the patient may present all the symptoms of tabes dorsalis. The course of my cases varied from two or three to about ten years, death supervening from some intercurrent malady.

The *Causation* is obscure. Syphilis was present in the history of some, but none of the cases was amenable to anti-syphilitic treatment.

Cervical Pachymeningitis (Hypertrophic Cervical Meningitis) is the same anatomical condition as the preceding, limited to the cervical region. It was first described by Professor J. M. Charcot. It is characterised by pain and atrophic paralysis in the arms from involvement of the sensory and motor roots, and subsequently by spastic paraplegia, due to descending lateral sclerosis from compression of the cord by the thickened membranes.

VIII. **Chronic System Lesions**.—Three degenerative diseases of the central nervous system of fairly common occurrence may first come under notice on account of weakness of the legs—disseminated sclerosis, general paralysis of the insane, and occasionally tabes dorsalis. Each has other and more characteristic symptoms, which are dealt with elsewhere. There are also five other rarer conditions, in which stiffness of the legs, due to lateral sclerosis, is a leading feature—primary lateral sclerosis (lateral sclerosis only), ataxic paraplegia (lateral and posterior sclerosis), amyotrophic lateral sclerosis (sclerosis of the lateral column and anterior horns), infantile cerebral and spinal paraplegia, and toxic sclerosis (see Figs. 148 and 150 in § 533).

IX. **Primary Spastic Paraplegia** (Synonyms : Primary Lateral Sclerosis, Primary Sclerosis of the Crossed Pyramidal Tracts, Erb's Paraplegia, Tabes Dorsalis Spas-

modique).—Lateral sclerosis on one or both sides is commonly met with as a spreading downwards from a localised disease in the brain or cord, but the primary spastic paraplegia consists of sclerosis of these columns without any primary disease above. Some deny the existence of a primary lateral sclerosis, and, personally, I regard the disease as extremely rare. (1) The onset is insidious, and the course is slow and painless, extending over many years. (2) Stiffness rather than absolute weakness is the leading symptom, and this gradually results in a stilted walk, till finally the patient walks on tiptoe without bending the knees. In advanced cases there is cross-legged progression, because the adductors are specially involved in the rigidity. Both legs are involved, though one may be a little worse than the other. Increased knee-jerk, ankle-clonus, and Babinski's sign are present as in other cases of lateral sclerosis. (3) Other features are negative—viz., no disturbance of sensation, no sphincter trouble, no bedsores, and no alteration in the electrical reactions. In the later stages the arms may become stiff.

Etiology.—The patients are usually from thirty to forty years of age. Most of the cases of so-called primary lateral sclerosis are early cases of disseminated sclerosis ; many of them are due to syphilitic meningo-myelitis. It is held by some that a genuine primary lateral sclerosis (similar to primary optic atrophy) may occur as a result of syphilis.

X. **Ataxic Paraplegia,** or postero-external sclerosis, is due to primary sclerosis in the posterior as well as the lateral columns. There is ataxic gait, but none of the other symptoms of locomotor ataxy. The knee-jerks are increased, and there is stiffness of the legs. This disease is met with chiefly in men of middle age, but much the same spinal lesions occur in Friedreich's disease—i.e., the hereditary ataxy of childhood. It is probable that most of these cases are instances of early disseminated sclerosis.

XI. **Amyotrophic Lateral Sclerosis** (Charcot) is due to disease affecting both the anterior horns and the lateral columns. The symptoms are usually confined to the arms for a time, and the disease is therefore described under Brachiplegia, § 603.

XII. **Infantile Cerebral and Spinal Paralyses** (including birth palsy, spastic diplegia, Little's disease).—The cerebral leg centres lie on the medial aspect just below the upper margin of each hemisphere, and during difficult or prolonged labour these are liable to meningeal hæmorrhage from compression. In other cases diffuse atrophy has been found, or porencephalus. When the condition occurs in prematurely born infants, it is believed to be due to defective development of the pyramidal tracts. Consequent on any of these lesions degenerative sclerosis takes place along the pyramidal tracts, and spastic paraplegia results. Nothing, perhaps, is noticed until the child begins, or ought to begin, to walk, which it is late in doing ; then the legs are found to be stiff, and signs of lateral sclerosis are present. The arms also may be involved—spastic diplegia. A limited athetosis or a more generalised chorea spastica may ensue. In all the varieties there is often a history of convulsions in infancy and a condition of mental deficiency. In other cases (hereditary spinal spastic paraplegia), however, the child is born with a stiffness of the legs, but without any mental symptoms or backwardness, and in these cases there has possibly been an injury to the cord at birth. Such patients may live bedridden to a considerable age—forty or fifty years. Some recognise a genuine hereditary form of the malady, which affects several members of a family, in whom some developmental defects may be assumed to exist. The feet are in a condition of talipes equinus, and spasm of the femoral adductors may lead to cross-leg progression.

XIII. **Toxic Combined Sclerosis.**—Sclerosis of the cord has been associated by the labours of Taylor,[1] Russell and others with various blood conditions, such as diabetes, pernicious anæmia, leukæmia, and pellagra. The sclerosis affects chiefly the posterior and lateral columns of the cervical and thoracic regions. The symptoms vary according to the column mainly affected, paraplegia is generally incomplete, and is sometimes

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[1] James Taylor, Roy. Med. Chir. Soc. Trans., 1895 ; J. R. Russell, the *Lancet,* 1898.

associated with ataxy and numbness. It may be diagnosed from similar spinal affections by the concurrence of one of the diseases mentioned.

Progressive stiffness of the legs, resulting in spastic paraplegia, is the leading symptom of **lathyrism** (lupinosis)—*i.e.*, poisoning by the use of meal derived from the seed of the chick pea (lathyrus savitus and l. cicera) mixed with other meal in the preparation of bread. The arms are very rarely affected. It is met with in India (chiefly), Algeria, and probably elsewhere. The underlying anatomical condition is not known, but it is probably a toxic sclerosis.

Treatment.—Resection of the posterior nerve roots [1] has been practised in cases of spasm due to disease of the upper neuron with a view of cutting off sensory stimuli from the periphery, but with doubtful benefit.

GROUP B. **The Lower Neuron or Flaccid Paraplegias** constitute a more limited group than the upper neuron paraplegias, and are distinguished from them by the four following features : (1) The paralysis is flaccid at any rate, for a considerable time ; (2) muscular wasting is a marked feature, and is attended by (3) characteristic electrical changes ; (4) the deep reflexes are absent. This group comprises :

✦ **§ 597. I. Multiple Peripheral Neuritis** (Synonyms : Multiple Neuritis, Polyneuritis) is a symmetrical toxic inflammation of the peripheral nerve trunks attended with pain and tenderness along their course, resulting in paresis and anæsthesia. This disease is more or less general in its distribution, but the paresis may predominate in the legs, and the patient frequently comes under our notice for paraplegia.

Symptoms.—The onset may be acute, subacute, or (more usually) chronic. Some observers make three varieties, according as the motor sensory, or ataxic symptoms predominate. In acute cases pyrexia may be present. (1) There is generally a premonitory stage in which there are numbness, tingling, cramps and twitchings with some weakness, in the legs or arms. Pain constitutes a prominent feature in all cases in which the sensory fibres are involved, the degree varying according to the acuteness of the process. The pain shoots along the course of the nerves, is symmetrical in distribution, and increased by movement. (2) There is also deep-seated tenderness along the nerve-trunks and in the muscle substance, especially in the calves. When (as in lead-palsy) the sensory fibres practically escape, pain and tenderness are absent. These symptoms are soon followed by (3) flaccid paresis and atrophy. The extensors are more affected than the flexors, and the patient has a characteristic foot-drop (or wrist-drop), and a difficulty in raising the foot in mounting stairs. (4) The electrical reactions vary, but there is usually

cases a diminution of faradic reaction. In many cases there is also a diminution to both galvanic poles (a condition which one does not obtain in anterior poliomyelitis), while in a few cases there is also typical Erb's reaction (§ 549), A.C.C. being greater than K.C.C. (5) The superficial and deep reflexes are diminished or disappear. (6) Anæsthetic and hyperæsthetic areas may, with care, be revealed. (7) Vaso-motor (redness, anasarca) and trophic (glossy skin, eruptions) changes may also occur Bladder and rectal troubles and bedsores are only met with at a very advanced stage, though in alcoholic cases with the dull mental state the excretions may be passed in bed. It is an interesting fact that the nerves involved vary somewhat with the toxic agent in operation. Thus, alcohol and beri-beri affect mainly the legs ; lead, the forearms ; diphtheria, the throat and neck muscles ; while arsenic appears in a few cases to affect mainly the trophic nerves of the skin. In lead paralysis the toxin selects the motor-fibres of the musculo-spiral nerve, and produces double wrist-drop. Accompanying severe alcoholic cases is loss of memory, apathy, and sometimes delirium.

Peripheral neuritis may have to be *diagnosed* from tabes. Thus in alcoholic neuritis there may be inco-ordination as well as the familiar pains and loss of deep reflexes (= pseudo-tabes). It is distinguished from it by the actual muscular weakness and the tenderness present, the high-stepping walk, and the absence of pupil changes. From poliomyelitis it is distinguished by the sensory changes and the age of the patient. The neuralgic pains may have to be differentiated from other causes of "pains in the limbs " (§ 452) ; and, lastly, the disease will often need to be distinguished from the other flaccid lower neuron pareses in this group, and from syringomyelia (§ 644). The acute febrile cases are hard to distinguish from Landry's paralysis and poliomyelitis in adults (§ 612).

Prognosis.—Multiple neuritis is essentially a chronic affection, but has seldom to be recovered from. The chief danger lies in the involvement of certain nerves, such as the phrenic and pneumogastric in diphtheritic paralysis, when sudden death may ensue. The acute febrile cases in a week from involvement of the respiratory muscles or cardiac paralysis, but a large proportion of cases of multiple neuritis get well in the course of three to twelve months, *if the cause in operation*, e.g., alcohol, be removed. Deformities from contractures may result in severe cases.

Causes.—Multiple neuritis may occur in either sex and at any age, but is most common in adults from twenty to fifty. Women are slightly more affected than men. There is always a morbid blood state in operation. The commonest of these in adults is alcohol ; in children, diphtheria. Other causes are : (a) Among the *hetero-toxins*—lead, arsenic, silver, ether, bisulphide of carbon and naphtha ; (b) among the *microbic*—influenza, tuberculosis, leprosy, enteric fever, variola (?), rheumatic, scarlatina, and other infectious fevers ; (c) among *auto-toxins*—gout, diabetes, cancer, oral sepsis, and pernicious anæmia. An attack has sometimes followed over-exertion or exposure. In beri-beri there is a

cause at work (see below). Traumatic cases come under the head of monoplegias.   o

Treatment.—The causes must be sought for and removed—*e.g.*, alcohol must be absolutely forbidden, and any gouty or other diathesis must be treated. Rest is the cardinal feature in all treatment, and merely remaining in bed will do much to cure. Prevent contraction of the paralysed limbs by sandbags. Iodide of potassium and quinine are of use for the elimination of toxins. When the acute and painful stage has subsided, galvanism is very valuable, and its systematic use with massage will hasten the restoration of the affected muscles.

§ 598. II. **Beri-Beri** frequently comes under notice for paraplegia, sometimes for dropsy of the legs. The paraplegic form is now recognised as due to a peripheral neuritis.

The *Symptoms* of beri-beri are of two kinds : (*a*) Those referable to the neuro-muscular system (the paralytic type) ; and (*b*) those referable to the cardio-vascular system (the œdematous type). Either form may occur alone, but usually the clinical picture is one of peripheral neuritis and cardiac failure, with dropsy, serous effusions and gastro-intestinal trouble. The onset may be sudden or gradual, with prodromata of languor, pains, and slight dyspnœa.

In (*a*) there is no œdema. The patient may be so thin and emaciated that the condition is called " dry beri-beri." The first symptom is a difficulty in walking, with the characteristic high-stepping gait of foot-drop. The knee-jerk is soon lost, and the muscular paralysis may extend from the legs to the arms, diaphragm, intercostals, and larynx. There is cutaneous anæsthesia, occurring first in the legs, and sometimes not spreading farther ; at the same time there is great muscular tenderness, especially noticeable in the calves. The sphincters are not affected. The heart is dilated towards the right, but cardiac symptoms play a minor part in this variety.

In (*b*) the characteristic symptoms are great dyspnœa and œdema, which is especially seen in the legs. Where dropsy is extreme the condition is called " wet beri-beri," the heart is dilated, especially to the right, with changing bruits from day to day. The digestion may remain unimpaired, and there is no fever.

Diagnosis.—Beri-beri has to be diagnosed from alcoholic and other forms of peripheral neuritis, and here the œdema and the constant involvement of the heart in beri-beri are important.

Prognosis.—In an epidemic the mortality may rise to 50 per cent., but normally only 10 per cent. of the cases end fatally. Death occurs usually from cardiac failure or asphyxia, and is thus often sudden and unexpected. Serious complications may arise in the form of œdema of the lungs or effusion into the serous cavities.

Causes.—Beri-beri is due to the absence of the vitamin which exists in that part of the rice grain which is removed by the polishing process. A vitamin is an essential part of a food-stuff without which its nutrient property is greatly impaired. Its constitution is unknown. Beri-beri occurs in Japan, China, the Malay Peninsula, and other parts where polished rice is much eaten. *Treatment* is symptomatic, and if symptoms of great venous obstruction occur with intense dyspnœa, bleeding must at once be resorted to. Prophylactic treatment consists in the avoidance of polished rice. Where this is impossible its injurious effects are apparently to some extent counterbalanced by the addition to the diet of meat and peas.

§ 599. **Other Causes of Flaccid Paraplegia.**—III. **Acute Anterior Poliomyelitis** at its onset may take the form of a paraplegia, but it more often affects a single limb ; in the course of a week or two it usually settles down into one limb, one segment of a limb, or one set of muscles. It occurs most frequently in children. It is described under Amyotrophy (§ 638).

IV. **Acute Transverse Myelitis** (§ 595) is at the outset a flaccid paraplegia, but the

paralysis is not atrophic excepting in the unusual event of the lesion occupying the cervical or the lumbar enlargements, which supply the arms and legs respectively.

V. **Landry's Paralysis** is also a flaccid paralysis (§ 612). It is a rare disease, resembling acute myelitis in some respects, though no gross lesions have been found *post-mortem*. There is a sudden onset of flaccid paralysis of the legs, followed by rapid extension to the muscles of the trunk and arms, and generally a rapidly fatal termination. An ultra-microscopic organism has been described in connection with the disease.

VI. **Syringomyelia** and **Intra-medullary Tumours** and **Hæmorrhage** may come under notice as paraplegia with flaccidity (§ 644).

GROUP C. **Functional or Variable Paraplegias.**—A destructive lesion of the motor tract gives rise to an unvarying paralysis which progresses steadily for better or worse ; but the leading feature of functional paraplegia is (1) that it tends to vary in intensity and sometimes in its other features from day to day ; (2) the loss of power is less marked—a paresis rather than a paralysis—and it does not clearly conform either to the upper or lower neuron type ; (3) it is accompanied by other evidences of the causal condition.

The principal varieties of variable paraplegia are—

　　　　　　　I. Hysterical paraplegia.
　　　　　　　II. Railway spine.
　　　　　　　III. Paraplegia after fevers.
　　　　　　　IV. Diver's paralysis.
　　　　　　　V. Reflex paralysis.

§ 600. **Hysterical Paraplegia** is the commonest, and may be taken as the type, of functional paraplegias. The nature of the lesion we do not know, and so variable are its clinical features that we have to rely mainly upon the fact of its occurrence in a person who is the subject of other hysterical manifestations. (1) The paraplegia is generally of sudden onset, and not infrequently dates from some hysterical seizure. I once saw a healthy-looking girl coming out of church suddenly fall down with functional paraplegia. It is rarely complete, and varies in intensity from day to day. Sometimes it is rigid, but almost as often flaccid, and the patient drags the foot in a characteristic way, resembling peripheral neuritis with foot-drop. (2) The paralysis and the other symptoms are inconsistent and evanescent. Thus, there may be hyperæsthesia in one leg, anæsthesia in the other, and the two may change places or disappear from day to day. All the symptoms may disappear rapidly, though I have observed cases of several years' duration.[1] (3) The muscles do not waste nor lose their electrical contractility. The deep reflexes may be unchanged, but are generally exaggerated, and spurious ankle-clonus may be present. The plantar reflex gives a normal flexor response showing the absence of organic disease of the spinal cord. (4) Incontinence of urine or fæces practically never occurs. (5) Other evidences of the hysterical diathesis are present. In the *Diagnosis* it is well to remember that the patient is practically always a female, though male cases have been recorded. There is an

[1] *Trans. Clin. Soc. Lond.*, vol. xxii., and the *Lancet*, 1901, vol. i.

absence of signs pointing to organic disease—much muscular atrophy, the R.D., ankle-clonus and Babinski's sign. These latter are regarded as absolutely conclusive of organic disease, but there is a kind of spurious ankle-clonus in many cases of hysteria which is only distinguished with difficulty from the ankle-clonus of organic disease. The *Causes* and *Treatment* are dealt with elsewhere (§ 557).

§ 601. II. **Railway Spine and other Functional Paraplegias.**—A severe shock or injury to the spine, such as occurs in railway accidents, may cause (1) a direct injury to the cord, e.g., hæmorrhage, laceration, or concussion ; (2) the shock may determine the occurrence of a degenerative lesion which does not come on perhaps for some months ; or (3) a form of painful paraplegia may ensue, not coming on until some days, weeks, or even months after the injury. It is with this last we are now concerned. Some, like Erichsen and Gowers, regard it as an incipient myelitis ; others, like Herbert Page, maintain that it is due to a functional change (? vascular). The symptoms consists of (1) severe spinal pain and tenderness (spinal neuralgia) and obscure peripheral sensations, such as tinglings, twitchings, or numbness in the legs. (2) A paresis or pseudo-paralysis, which Page believes may be due to the fear of pain produced by movement. (3) Bladder symptoms are sometimes present, such as frequency of micturition or a dribbling at the end, or a difficulty in defæcation. These cases are very difficult to treat successfully especially when pecuniary compensation is (subconsciously) anticipated. Psychotherapy and rest, with change of scene and occupation, are the main indications in treatment.

III. **Paraplegia after Fevers.**—The paraplegia following beri-beri and most fevers is due to peripheral neuritis, or myelitis, but that which is especially liable to occur after jungle fever is held by Indian authorities to be due to anæmia of the cord All these forms are characterised by the paralysis being incomplete and usually transient.

IV. **Diver's Paralysis** (Synonyms : Caisson Disease, Compressed Air Illness, is a paraplegia which occurs in men who work under water, or tunnel under the ground at high atmospheric pressure. It is due not so much to the pressure the men are subjected to as to the sudden release of that pressure, which produces congestion of the cord, with liberation of gas from the blood. In fatal cases hæmorrhage and myelitis have been found. This was well illustrated in a case shown by Dr. Robert Maguire at the Medical Society.[1] The patient had had six or seven previous attacks, and the last was brought on by his sudden rush to the surface from a depth of 150 feet —that is to say, from a pressure of 90 pounds to the square inch to one of 15 pounds to the square inch. The paresis comes on suddenly when the patient returns to the normal atmospheric pressure. It is never quite complete. It usually affects the legs, rarely the arms to any extent. Anæsthesia, severe pain, and sphincter paralysis only occur in the graver cases. Auditory vertigo, hæmorrhage from the nose, lungs, and other parts sometimes occur. The prognosis is favourable in most cases ; pain and paresis pass off in a few days to six weeks. A few cases have died.

Treatment.—Curative measures consist simply of rest and the avoidance of alcohol —a failing to which most of these workmen are addicted. Ergot has been recommended. Preventive treatment consists in adopting precautions for gradual decompression. Men employed in diving or in tunnel working under pressure should be compelled to undergo a very gradual process of decompression. Dr. Snell recommends ten minutes' decompression for each atmosphere of pressure.

V. **Reflex Paraplegia** is a variety which some do not admit. It was first described by Trousseau. After operations on the anus there may be weakness of the legs or inability to pass urine for days. The loss of power appears to be never quite complete. The only means of identification consists in the presence of a reflex cause and the disappearance of the paralysis when this is removed. Probably most cases supposed to be of reflex origin are cases of neuritis ascending to the cord.

§ 602. The Prognosis and Treatment of Paraplegia.—*General Remarks on the Prognosis of Paraplegia.*—Paraplegia is always a serious symptom, not only because it prevents locomotion, but because it indicates structural or functional disease of that important structure the spinal cord. In general terms (1) all functional disorders are more favourable than organic ; (2) flaccid paraplegias tend to run a quicker course either towards recovery or death than rigid paraplegias ; (3) the most unfavourable signs are bedsores, and the implication of the bladder and rectum. The different forms of paraplegia may be grouped for the purposes of prognosis into four groups (see table below). The presence of complications adds to the gravity of any case. Many cases die of cystitis and pyelitis or the chronic septicæmia which results from bedsores ; others from pneumonia or other complications. Finally, efficient or inefficient nursing is an extremely important factor in the prognosis, for these cases constitute the test and trial of all that makes for efficiency in nursing.

The *Treatment of paraplegia*, excepting in " functional " cases, is not very hopeful, for a seriously damaged portion of the cord can never be fully restored. There are three indications : To remove the cause, to prevent complications, and to restore the functions (*a*) To remove the cause: (1) Anti-syphilitic treatment—salvarsan,

·PROGNOSIS OF THE DIFFERENT FORMS OF PARAPLEGIA.

Those which generally recover completely, either spontaneously or under adequate treatment.	*Those which generally terminate in* PARTIAL *recovery, either spontaneously or under adequate treatment.*	*Those which tend to become chronic and incurable, but not fatal.*	*Those which tend to a fatal issue.*
Hysterical and the other varieties of functional para-plegia. Peripheral neuritis. Some cases of Pott's disease. Non-malignant extra-medullary tumours. Paraplegia after fevers.	Most cases of Pott's disease. Anterior poliomyelitis.	Dorsal myelitis. Embolism. Some spinal injuries. Chronic spinal meningitis. Hæmorrhage. Primary lateral sclerosis. Ataxic paraplegia. Amyotrophic lateral sclerosis. Infantile cerebral and spinal paraplegia.	Malignant and inaccessible tumours. Acute cervical or lumbar myelitis. Some cases of hæmorrhage. Landry's Paralysis. Malformation.

mercury or iodide of potassium, should be employed at once in all possibly syphilitic cases. If inflammation or congestion of the cord be suspected, belladonna and ergot be given ; experimentally these remedies produce contraction of the vessels of the pia mater. Rest and the prone position, the patient lying on the stomach, are of advantage. Where hæmorrhage is suspected, prolonged absolute rest in the prone position is necessary. (3) Counter-irritation, I believe, might with advantage be used more often than it is, especially in the upper neuron group—blisters to the spine, frequent cupping, hot douches, frictions, mustard or ung. iod. rubbed into the spine. In chronic cases the best treatment, in my experience, is the " coup de fer " of the French—that is to say, dotting the point of a Paquelin thermo-cautery down the spine every other day. Several cases of sclerosis under my care have derived advantage from these methods. (4) Surgery comes to our aid in cases of Pott's disease, injury, malformations, and some tumours. Certain of the causes require special treatment. In *Pott's disease* rest in a prone or supine position affords a good chance of complete recovery (head and arms fixed to the wall, counter-extension by weight). In older people laminectomy may be performed at once because the disease tends in them to progress. Sayre's jacket and various kinds of apparatus are used ; one of the best of these, perhaps, is one designed by Mr. Jackson Clarke.[1] In *acute myelitis*

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[1] See discussion Clin. Soc., the *Lancet,* March, 1900.

an ice-bag to the spine and the administration of atropine subcutaneously and locally have been advocated, though but little can be done beyond rest and the prevention of bedsores. For *chronic meningitis* (pachymeningitis) and the upper neuron scleroses (see §§ 592–596), the iodides or atropine may be tried, but counter-irritation is the best. The treatment of *hysterical paraplegia* is given elsewhere; the value of faradic and static electricity is undoubted.

(*b*) To prevent complications should be our endeavour in all cases. A catheter carefully *sterilised* should be passed three or four times in the twenty-four hours in cases of partial or total retention, and with the utmost care, for the parts are often anæsthetic. The patient should be placed upon a water-bed, the bowels kept gently acting with laxatives or enemata ; and the formation of bedsores prevented by clean-liness, dryness, and the relief of pressure at all prominent points. Good nursing is of the highest importance in all of these cases, especially in cases of myelitis.

(*c*) The restoration of muscular function may be promoted by the application of galvanism, faradism (particularly the combined current), massage, and passive move-   ments. These means are more useful in flaccid paraplegias, and are not suitable in recent or irritable cases or in the earlier stages of acute myelitis. Nerve tonics, strychnine, phosphorus, arsenic, cod-liver oil, iron, quinine, are all of value to promote nutrition.

The patient complains of weakness or **paralysis** *of* BOTH ARMS. The case is one of BRACHIPLEGIA (*i.e.*, Brachial Diplegia).

§ **603.** **Brachiplegia** is paralysis of both arms without paralysis of the legs. It is not a very common condition, and must not be confused with diplegia, which is double hemiplegia, nor with brachial monoplegia. It is met with occasionally in the subjoined diseases. In the *Diagnosis of the cause* you should first ascertain whether you have to do with a *lower* neuron lesion, an *upper* neuron lesion, or a combination of both.

I. Double *musculo-spiral paralysis* (p. 859) is the commonest cause of brachiplegia, as met with in lead poisoning and sometimes other toxic conditions, such as poisoning by arsenic or silver.

II. In *syringomyelia* (§ 644) wasting and weakness of one or both arms may be the earliest feature. The paralysis is usually flaccid, but may be rigid, and is accompanied by loss of temperature and pain sense with retention of tactile sensibility.

III. In a few cases of *acute anterior poliomyelitis* (§ 638) both arms may be paralysed at the same time.

IV. In certain cases of *idiopathic muscular atrophy* (*primitive myopathy*), weakness of the muscles of the arms and shoulder girdle may be the earliest, and for a long time the only symptom.

V. *Injury to the cord* in the cervical region causing hæmorrhage into the anterior horns may result in atrophic paralysis of the arms.

VI. *In hypertrophic cervical meningitis* and in *extra-medullary tumour* in the region of the cervical enlargement there is acute pain in the arms, and the " claw hand " usually develops.

VII. **Amyotrophic lateral sclerosis** (Charcot) is a somewhat rare disease, due to disease of both the anterior horns and the lateral columns. In the first stage, lasting usually from four to twelve months, the symptoms are mostly confined to the upper extremities, which gradually *undergo atrophy*. At the same time they show a tendency to rigidity and to an increase of the deep reflexes. Both arms are usually involved, but not infrequently one side more than the other. In the second stage the lower extremities become invaded and symptoms of Primary Lateral Sclerosis supervene. In the third stage the bulb is involved, the patient dying from bulbar paralysis. The disease differs from progressive muscular atrophy in the presence of the stiffness both of the arms and of the legs, and also in its relatively rapid course, for death generally

takes place in from one to three years. It more closely resembles cervical pachy-meningitis, excepting in the absence of pain.

• *The patient complains of weakness or* **paralysis** *in* ONE ARM *or* ONE LEG. The case is one of MONOPLEGIA.

§ 604. **Monoplegia** is loss of power in one limb. Monoplegia brachialis is paralysis of one arm ; monoplegia cruralis, of one leg. Its causes are as follows :

(*a*) *Single Nerve and Plexus Paralysis* (§ 605).

• I. Single nerve paralysis.
  II. Plexus paralysis.
  III. Occupation neuroses.
  IV. Arthritic atrophy.

(*b*) *Spinal Monoplegias* (§ 607).

  I. Acute anterior poliomyelitis.
  II. Chronic anterior poliomyelitis.
  III. Spinal tumours, Pott's disease, and other causes of paraplegia (occasionally).
  IV. Syringomyelia.
  V. Amyotrophic lateral sclerosis, cervical pachymeningitis and other causes of brachiplegia (occasionally).

(*c*) *Cerebral Monoplegias* (§ 608).

  I. Focal cortical lesions.
  II. Hysterical monoplegia.

(*d*) *Certain Primitive Myopathies* (§ 640).

The chief POINTS TO INVESTIGATE are, first, precisely which of the muscles are affected ; secondly, the electrical reaction ; thirdly, the sensation of the affected part ; and fourthly, the presence of any source of pressure along the course of the nerve trunks, plexuses, or roots. It is often difficult to decide whether an alleged weakness in the forearm is not in reality due to disease in one of the smaller joints. In any case the joints should be examined, because even after slight injury an arthritic amyotrophy (see below) occasionally ensues.

Monoplegia may be due to a lesion situated (*a*) in the peripheral nerves, or the nerve-plexuses ; (*b*) spinal affections involving the anterior horns or anterior roots ; or very rarely (*c*) localised cerebral lesions.

605. Single Nerve Paralysis.—Peripheral nerve and nerve-plexus lesions give rise to a monoplegia which, like all lower motor neuron lesions, is *flaccid, accompanied by R.D.*, and followed by *atrophy* of the affected muscles. The key to the detection of the nerve involved consists in the identification of the precise muscles affected. The lesion may be an injury, pressure, or inflammation, but in each case the symptoms are much the same in kind, though they differ somewhat in degree. A divided nerve may be taken as the type.

Symptoms.—Division of a *motor* nerve (or its severe contusion or compression) gives rise to immediate (1) flaccid paralysis of the muscles supplied, and alterations in the electrical reactions ; (2) abolition of the tendon reflexes in the muscular region involved—followed in the course of the ensuing week by (3) muscular atrophy ; and (4) total loss of contractility to faradism. Most nerves are. *mixed,* and pain and various kinds of

paræsthesia are very constant; indeed, pain is the most prominent symptom in acute inflammation or severe injury. Anæsthesia is much less constant. Sensation may be retained, even after complete section of a sensory nerve root, owing to the extensive overlapping of the nerve areas, and the like is true of the smaller nerves, not, however, of such a nerve as the median. Sensation, when lost, is sooner recovered than motion. Vaso-motor and trophic disorders often result—redness, blueness, hyperidrosis, œdema, wasting of the skin (glossy skin), subcutaneous tissue and bones, and vesicles followed by badly healing sores.[1]

The *Diagnosis* is greatly aided by the electrical reactions. Thus we are enabled to distinguish a nerve injury from (1) a direct muscular injury; (2) arthritic amyotrophy following, *e.g.*, a slight contusion of a joint; and (3) spinal monoplegias which are further distinguished by the different course they run, and the associated symptoms (§ 607).

The *Prognosis* depends upon the nature and degree of the lesion. Com plete R.D. indicates complete severance of a nerve. But it is won k rful how a nerve will repair with rest, as Mr. John Hilton [2] long ago pointed out.

The *Causes* of peripheral nerve and plexus lesions are manifold, but may be grouped under two headings. The special causes affecting particular nerves will be considered afterwards.

(*a*) *Injury or Pressure.*—Pressure during sleep (especially after alcoholic intoxication), ligatures, the use of crutches (crutch-palsy of the musculo-spiral), luxation of the humerus or other bones, bullet wounds, stabs, and direct blows (*e.g.*, on the brachial plexus above the clavicle), or fractures, may injure the nerve; callus (recognised as a cause by the pain coming on some time after fracture) may involve or compress a nerve, or the nerve may be included in cicatricial tissue. Sudden extension of the arm upwards may lead to severe damage or laceration of the brachial plexus, and the child may suffer similar damage during parturition; severe muscular action (*e.g.*, contraction of the triceps on the musculo-spiral) may cause damage (as Sir William Gowers has shown) by compression. Disease of the bones (*e.g.*, caries or syphilitic disease), or enlarged glands or other tumours may lead to compression, and extensive pleurisy at one apex may have the same effect. Injury may lead to ascending neuritis.

(*β*) A *Toxic Agent* (as already mentioned in Multiple Neuritis) sometimes acts as a predisposing factor, sometimes as the sole cause of nerve paralysis; and the toxic agent often seems to have an unexplained proclivity for certain nerves, as in the case of lead for the musculo-spiral. Alcohol generally produces multiple neuritis, but may act as a contributory factor to slight injury or local compression. Arsenic, and occasionally mercury and silver, have been known to produce musculo-spiral paralysis. Diphtheria and influenza are well-known toxic causes of neuritis, and the other infectious fevers may act similarly. Diabetes and

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[1] See a case reported by the author in *Brain*, part lxiii., 1893.
[2] Hilton's "Rest and Pain," edited by Jacobson; London, Bell and Sons, 1887.

enteric fever have been accompanied by paralysis of peripheral nerves. Paralysis of the musculo-spiral has been observed in enteric fever, articular rheumatism, and pregnancy ; and rheumatism and gout are believed to operate similarly.

In the *Treatment* of injury or compression of a nerve the main object is to ascertain and remove the cause. Rest is of paramount importance. Weak galvanism (6 to 8 ma.) regularly applied, especially in the form of a limb bath, is of value, and later this may be combined with faradism and massage. If at the end of many months' perseverance the R.D. is still present, surgery may be summoned to our aid. Surgical treatment will sometimes completely restore muscles that have been paralysed for many years. The distal end of an injured or diseased nerve has been successfully spliced on to another healthy nerve. The treatment of single nerve lesions is also referred to under Peripheral Neuritis (§ 597) and Neuralgia (§ 641).

The SYMPTOMS and CAUSES of PARALYSIS OF INDIVIDUAL NERVES will now be considered. For sensory symptoms, see Figs. 162 to 165.

The *musculo spiral* nerve is paralysed more frequently than any other nerve of the extremities, owing to its peculiar course and superficial position, and paralysis of it may be described as a type of localised nerve paralysis. It may be involved in paralysis of the brachial plexus (*vide snfra*). The *Symptoms* are wrist drop, due to paralysis of the extensors ot the wrist and of the fingers, together with the supinator longus and brevis. Sensation is affected only in a few severe cases. The commonest *Cause* is pressure or injury of some kind (group *a*). A common form of paralysis of the musculo-spiral nerve is the so-called pressure palsy from lying on one arm. It is predisposed to by chronic alcoholism. Lead poisoning is also a very common cause of paralysis limited to the musculo-spiral nerve, the supinator longus, however, generally escaping.

The OTHER NERVES are less frequently involved singly. The symptoms are given in a table below. The principal causes are as follows :

Circumflex.—Injury to shoulder, pressure, toxic (diabetes, lead).

Long Thoracic.—Over-exertion, lifting heavy weights, injury, etc.

Median—Traumatism, occupation neuroses (dentists, joiners, cigar-makers, etc.).

Ulnar —Traumatism, occupation neuroses. Injury or disease of the lowest part of the cervical enlargement may involve only the fibres going to the ulnar nerve ; this should be remembered before diagnosing a case as ulnar paralysis. Numbness and anæsthesia in this area occur in tabes dorsalis.

Anterior Crural.—Pressure by pelvic or vertebral tumours ; toxic (gout, alcohol, diabetes) ; femoral aneurysm.

Great Sciatic.—Pelvic tumours and inflammation, injury to femur, compression during parturition. Pressure within the pelvis often involves the peroneal fibres only. An habitually loaded rectum may cause paresis of the muscles of, and pain in, the left leg, but it should be remembered that when sciatica is attended with marked atrophy, the question of cancer of the sigmoid flexure, or rectum, or some other pelvic tumour should be considered. Toxic causes include gonorrhœa, gout, rheumatism, fevers, nephritis.

Phrenic Nerve.—Diphtheria, injury or disease of the third and fourth cervical roots. The symptoms of paralysis of the phrenic nerve are (1) dyspnœa on exertion ; during deep inspiration the abdomen does not protrude, owing to paralysis of the diaphragm.

§ 606. II. **Plexus Paralysis,** paralysis due to a lesion involving a nerve plexus, is another cause of monoplegia. It is recognised by the number and extent of the muscles involved. It is not always possible to distinguish nerve root from nerve trunk involvement.
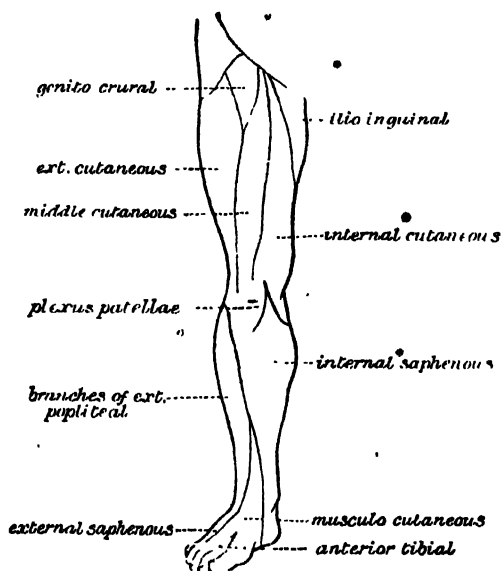
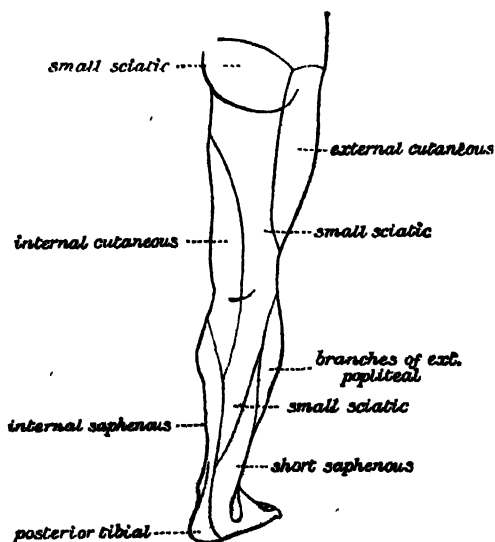FIG. 162.—Approximate areas of CUTANEOUS SENSATION supplied by the peripheral nerves.

FIG. 163.—Approximate areas of CUTANEOUS SENSATION supplied by the peripheral nerves.
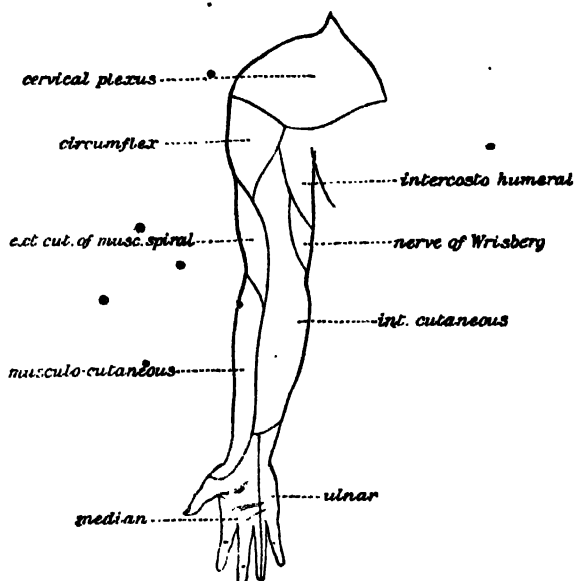
FIG. 164.—Approximate areas of CUTANEOUS SENSATION supplied by the peripheral nerves.
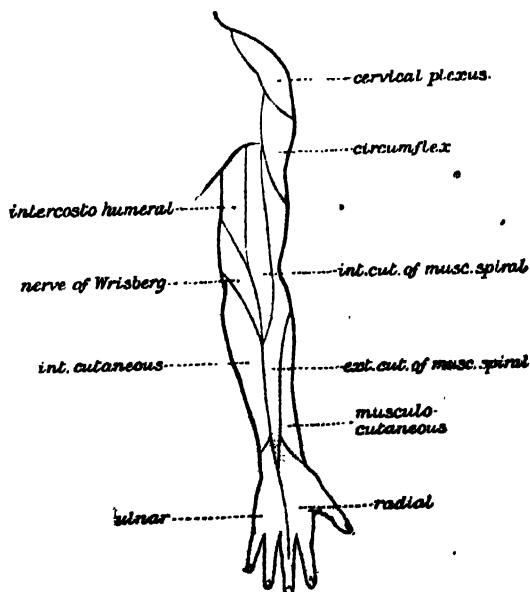

FIG. 165.—Approximate areas of CUTANEOUS SENSATION supplied by the peripheral nerves.

I. There are three forms of **brachial plexus paralysis** :

(1) *Upper brachial plexus paralysis* of the shoulder and arm (Duchenne-Erb) involves the muscles of the upper arm—namely, the deltoid, biceps, brachialis anticus and supinators, sometimes the infra-spinatus and sub-scapularis. It results from tumours or lesions involving the fifth and sixth cervical roots, or from injury causing pressure of the clavicle against the first rib or spinal column.[1]

(2) *Lower brachial plexus paralysis* of the forearm is much rarer. It involves the muscles of the hand and the flexors of the forearm, sensation being impaired in the region of the ulnar nerve, the inner surface of forearm and upper arm.

(3) *Total brachial plexus paralysis* is very rare, and always traumatic.

Brachial neuritis, a toxic, ill-defined affection of several of the nerves of the arm, or of the whole of the brachial plexus, is described under Pain (§ 641), which is its leading symptom.

A. **Cervical rib** gives rise to symptoms of numbness and pain along the inner side of the arm, with diminution of sensation over the third and fourth fingers. Muscular atrophy ensues, affecting chiefly the hypothenar and thenar eminences and the interossei. The limb may be cold and blue, and the pulse hardly palpable. The diagnosis from syringomyelia and progressive muscular atrophy is revealed by X-ray examination.

Obstetrical paralysis, due to injury during parturition, very often takes the form of upper brachial plexus paralysis; sometimes the facial nerve is injured by the forceps. Recovery generally takes place in course of time. Other forms of birth palsies are referred to under Hemiplegia.

B. **Lumbar and sacral plexus paralysis** is much rarer than brachial, and is due to tumours or disease adjacent to the lumbar or sacral vertebræ. The sigmoid flexure is a frequent primary seat of malignant disease, and may give rise to sacral or sciatic paralysis. The anterior crural nerve (see table) is generally involved.

III. **Occupation neuroses**, such as writer's cramp, telegraphist's palsy, etc., may be attended by paresis, but they more often consist of a muscle spasm, under which they are described (§ 620).

IV. **Arthritic amyotrophy** is described under Amyotrophy (§ 640). It is due to wasting of some of the extensor muscles following contusion or disease of the joint, sometimes of quite a trivial kind.

§ 607. Spinal Monoplegias.—Affections of the spinal cord may give rise to monoplegia when the lesion involves the anterior horns or anterior roots. We are then in the presence of a *lower motor neuron paralysis*, which differs from the last group only in the circumstances under which the paralysis occurs, and in the distribution of the muscles involved.

I. **Acute Poliomyelitis** (Infantile Paralysis) is described under Muscular Atrophy (§ 638). It is the commonest cause of monoplegia in childhood, and though it may affect more than one limb at the onset, 55 per cent. of all cases settle down into a crural monoplegia. In 23 per cent. of the cases one arm is affected.

II. **Chronic Anterior Poliomyelitis** (Progressive Muscular Atrophy) is met with chiefly in adults as a slow progressive amyotrophy starting at the ends of the limbs, generally in the hands, sometimes in one hand.

III. **Spinal Tumours**, or **Pott's Disease**, especially when pressing on the nerve roots in the region of the brachial or lumbar plexus, may commence with monoplegia, are recognised by lancinating pains shooting from the back down the limb, the gradual

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[1] It was a case of paralysis of the upper brachial plexus type that Harris and Low successfully treated by cross union of the nerve roots (*Brit. Med. Journ.*, October 24, 1903).

TABLE OF ACTIONS OF MUSCLES AND THEIR NERVE SUPPLY.

Nerve.	Muscles Supplied.	Defective Movement.	Deformity Produced.
Posterior thoracic or external respiratory nerve of Bell.	Serratus magnus.	Arm cannot be raised above a horizontal position.	Scapula higher than normal. On attempting to thrust arm forward scapula lifts itself with its inner border "wing-like" from the thorax.
Supra-scapular.	Supra- and Infra-spinatus.	Disturbance in function not pronounced, paralysis of infra-spinatus produces difficulty in rotating the arm; of supra-spinatus, fatigue in lifting arm.	Scapular spine prominent when infraspinatus atrophied.
External anterior thoracic	Pectoralis major (upper part).	No movement entirely prevented, but adduction of arm imperfectly performed.	
Internal anterior thoracic.	Pectoralis major and Pectoralis minor.		
Musculo-cutaneous.	Coraco-brachialis. Biceps. Brachialis anticus.	Forearm flexed with difficulty, especially in supinated position.	Characteristic depression on outer surface of upper arm between insertion of deltoid and origin of supinator longus.
Subscapular.	Subscapularis. Teres major. Latissimus dorsi.	In paralysis of latissimus dorsi forcible backward depression of raised arm is lost. In paralysis of teres major elevation of shoulder with the arm against side is lost.	
Circumflex.	Deltoid. Teres minor.	Arm cannot be abducted nor elevated backward or forward.	Change in the shape of the shoulder; relaxation of shoulder-joint develops later.
Musculo-spiral.	Triceps. Anconeus. Supinator longus. Extensor carpi radialis longior. Brachialis anticus.	Elbow, wrist, and basal phalanges of fingers cannot be extended; grip weakened; impaired flexion of forearm if supinator longus is involved.	"Wrist-drop," fingers flexed in metacarpo - phalangeal joints; thumb opposed to fingers and somewhat depressed downwards.
Posterior interosseous branch.	All the supinators and extensors of carpus and fingers except supinator longus and extensor carpi radialis longior.		Supinator longus paralysis detected by placing forearm midway between pronation and supination, when flexion against resistance does not bring muscle belly into view.

TABLE OF ACTIONS OF MUSCLES AND THEIR NERVE SUPPLY
—*continued*.

Nerve.	Muscles Supplied.	Defective Movement.	Deformity Produced.
Median.	Pronator radii teres. Palmaris longus. Flexor carpi radialis. Flexor sublimis digitorum. Flexor longus pollicis. Opponens pollicis. Abductor pollicis. First and second lumbricales. Parts of flexor brevis pollicis and flexor profundus digitorum.	Flexion of hand with slight force and with ulnar deviation. Fingers cannot be properly flexed at first phalangeal joint, while flexion of terminal phalanges only practicable in last three fingers. Pronation of arm lost. Opposition and flexion of terminal phalanx of thumb lost.	Position of hand not markedly altered, generally turned towar ulna and held tightly supl- Wasting of th muscles con cuous.
Ulnar.	Flexor carpi ulnaris. Adductor pollicis. Muscles of ball of little finger. Interossei. Last two lumbricales. Part of flexor brevis pollicis and flexor profundus digitorum.	Patient can flex hand, but only with adduction towards radius. Inability to flex terminal phalanges of last three fingers and to adduct thumb. Basal phalanges cannot be satisfactorily flexed nor middle and dista phalanges extended. Abduction and adduction of fingers impossible.	"Claw hand," most pronounced in fourth and fifth fingers. First phalanx in extreme extension and second and third held firmly flexed; atrophy of hypothenar eminence and of interossei.
Intercostals.	Intercostals. Rectus abdominis. External oblique. Internal oblique. Transversalis.	In paralysis of abdominal muscles forced expiration interfered with; only possible to rise from a reclining position by using arm as a support.	Lordosis; pelvis strongly bent forward; abdomen and nates show up prominently.
Lumbar.	Erector spinæ. Quadratus lumborum.	The back in walking and standing thrown backward; on sitting spinal column is arched convexly backward.	Slight lordosis which disappears re- clining; pelvis raised.
Anterior crural.	Sartorius. Pectineus. Quadratus femoris.	Inability to extend lower leg. Absence of knee reflex. Paralysis of ilio-psoas evidenced by inability to flex hip.	Gait disturbed. Patient stepping carefully, avoiding flexion of knee.
Obturator.	Gracilis. Obturator externus. Adductor longus. Adductor brevis. Adductor magnus.	Adduction and to a slight extent external and internal rotation impaired.	

TABLE OF ACTIONS OF MUSCLES AND THEIR NERVE SUPPLY — *continued*.

Nerve.	Muscles Supplied	Defective Movement.	Deformity Produced
Inf. gluteal	Gluteus maximus.	Abduction and particularly extension at hip joint hampered	The "walking, leg swings too far inwards also excessive lifting and sinking and forward tilting of pelvis
Superior gluteal	Gluteus medius. Gluteus minimus. Tensor vaginae femoris	Loss of abduction and circumduction of thigh	
Internal popliteal	Gastrocnemius Soleus Tibialis posticus Flexor communis digitorum Flexor longus hallucis	Loss of plantar flexion of foot and toes Patient unable to lift himself upon tips of his toes Walking difficult	Claw position of toe (pied en griffe), pes calcaneus or valgus
External popliteal	Tibialis anticus Extensor proprius hallucis Extensor longus digitorum Peronei Extensor brevis digitorum	Foot falls from its own weight, and cannot be raised, nor can first phalanx be extended Walking difficult, toes scrape the floor	"Foot-drop." Foot remains in equinovarus position

advent and prolonged course of the paralysis, and the concomitant symptoms due to pressure upon the cord (§ 593).

IV. Syringomyelia frequently starts as an atrophic paralysis of one, sometimes of both arms, associated with various sensory and trophic symptoms (§ 644).

V. Amyotrophic Lateral Sclerosis and Cervical Pachymeningitis commence as an atrophic paralysis of one or both hands. They are relatively rare diseases.

§ 608. Cerebral Monoplegia.—Cerebral lesions causing paralysis generally produce hemiplegia; only very rarely a monoplegia. The nearer the lesion to the motor cortex the more likely is it to produce a limited paralysis. The clinical features here are those of an upper motor neuron lesion, which would at once distinguish it from the two preceding groups.

I. Real cortical lesions producing monoplegias are comparatively rare. Embolism or thrombosis of the anterior cerebral artery (not a common position) produces a crural monoplegia. Occasionally vascular lesions in the cortex involve the arm and face centres only. Tumours exactly limited to one of these centres are practically unknown. Several cases of cerebral monoplegias as the result of war injuries have recently been recorded.

II. Hysterical Monoplegia (arm or leg) is more common than organic cerebral monoplegia. Hysterical monoplegia generally dates from an accident, or nerve seizure, and it is usually attended by segmental anæsthesia (in which the area of loss of sensation is bounded by a circular line drawn round the limb, usually at a joint).[1]

The patient complains of a **widespread paralysis** *or muscular weakness.* The case is one of GENERALISED PARALYSIS (general debility being excluded).

[1] A typical case forms the subject of a clinical lecture by the author in the *Clinical Journal*, May, 1904.

§ 609. Generalised Paralysis is met with towards the end of quite a number of nervous diseases, but only a few begin with an involvement of all the limbs. We must be careful to exclude cases of general debility (Chapter XVI). General paralysis accompanied by stiffness is described under Spasm (§ 618).

Toxic or Functional.

Multiple peripheral neuritis due to alcoholism, diphtheria, etc.
Hysteria.
Paralysis agitans.
Myasthenia gravis.
Family periodic paralysis.

Intracranial Lesions.

General paralysis of the insane.
Diffuse basal tumours.
Cerebellar tumours.
Cerebral pachymening.
Infantile diplegia cerebralis.

Spinal and Bulbo-Spinal Lesions.

Disease or injury high up in the spinal cord.
Cervical pachymeningitis.
Disseminated sclerosis and other chronic degenerative conditions.
Landry's paralysis.
Diffuse myelitis.
Acute anterior poliomyelitis.
Progressive spinal muscular atrophy of infants.

Amyotrophy.

Idiopathic muscular atrophy.
Amyotonia congenita.

In seeking to DIAGNOSE THE CAUSE of a case of generalised paralysis one naturally turns *first* to some possible *toxæmic* or *functional* condition such as alcoholism and hysteria.

Secondly, the possibility of some *intracranial disease,* such as a basal cerebral or cerebellar tumour encroaching upon the brain-stem, or general paralysis of the insane would next engage our attention, and we should seek for the associated symptoms.

Thirdly, we might suspect some *spinal* or *bulbo-spinal* disease when the peripheral symptoms, or symptoms referable to the cranial nerves, would be more prominent than the cerebral or mental symptoms.

In the last stages of **hydrophobia** generalised paralysis ensues.

In **Hysteria** I have occasionally met with a flaccid paralysis coming on suddenly and affecting all the limbs.

Paralysis Agitans (§ 627) from the outset attended by weakness of all the limbs. This gradually increases until the patient becomes bedridden, and all the limbs become stiff and powerless.

§ 610. Myasthenia Gravis (Asthenic Bulbar Paralysis) is a rare condition, possibly due to a toxin affecting the end organs of the voluntary motor nerves. It consists of progressive fluctuating weakness of all the cerebro-spinal muscles. It was first described by Willis in "The London Practice of Physic" in 1865, and was studied again by Sir Samuel Wilks in 1877, and Erb in 1878. About sixty cases had been recorded up to 1900.

Symptoms.—1. The patient easily becomes tired. The first thing in the morning the muscles may contract normally, but after a little exercise they get weaker, and .they are worst in the evening. •All the voluntary muscles of the body tend to be affected, but especially those of the face, eyes, and neck. Bilateral ptosis is present in about 80 per cent. of the cases, and all the extrinsic ocular muscles are weak, while the intrinsic muscles escape. The face is expressionless, and there is often a difficulty in swallowing and in articulation, similar to that observed in bulbar paralysis. A nasal speech after talking awhile and a difficulty of mastication after starting to eat are other instances of the rapid fatigue of the muscles. Paroxysmal dyspnœa may supervene. The symptoms vary in intensity from time to time, and may even temporarily disappear, but the tendency is for the patient to get gradually weaker. 2. The reaction to faradism is characteristic. Although the muscles contract at the moment when the terminal is first applied, they soon become completely flaccid again, and after several applications may fail altogether to respond, while they continue to respond to galvanism (myasthenic reaction). ˚3. Fibrillation, obvious muscular atrophy, reaction of degeneration, and sensory changes are all absent.

Diagnosis.—Cases may be mistaken for bulbar paralysis, in which, however, the ocular symptoms are wanting. In Addison's disease pigmentation, emaciation, and other symptoms are present.

The *Prognosis* is grave, the disease is insidious, and may last for several years, but ends fatally, either from asphyxia or some intercurrent affection.

The *Etiology* is uncertain. Young persons are mostly affected. All that we can say of the causation is that the disease is due to some peculiar blood state. The thymus is often found to be persistent and enlarged, and small masses of lymphoid tissue may be found in the muscles, notably in those supplied by the cranial nerves. Dr. Farquhar Buzzard has described minute lymphorrhages in the muscular-tissue as a constant and characteristic lesion. The carbohydrate metabolism of the muscles is defective and creatinuria is common.

For the *Treatment* rest, massage, and full doses of strychnine are indicated.

§ 611. **Family Periodic Paralysis** is a rare disease characterised by attacks of paralysis of gradual onset after exertion. The attacks last six to sixty hours, and are attended by motor flaccid paralysis of limbs and trunk, with absence of response to galvanism or faradism. The left ventricle becomes temporarily dilated during the attack. Here again a morbid condition of the blood is doubtless at work. Acetonuria may be present. Treatment with diuretics and alkaline drinks is indicated.

INTRACRANIAL LESIONS mostly produce hemiplegia or paralysis of cranial nerves, but certain of them may produce generalised paralysis. **General Paralysis of the Insane** (§ 576), in which the cerebral cortex is diseased, culminates in a generalised paralysis.

Diffuse basal tumours, or gummatous meningitis involving the pons or the peduncles, may cause generalised paralysis, but in such cases the cranial nerves are also involved.

Cerebellar tumours by pressing on the brain-stem may produce generalised paralysis, associated with nystagmus and a characteristic gait (§ 613).

Cerebral pachymeningitis produces a vague generalised paresis and mental weakness.

Hæmorrhage or injury at birth may cause paralysis of both arms and legs (infantile spastic diplegia).

Among SPINAL and BULBO-SPINAL LESIONS **Pott's disease** and spinal tumours high up in the cord may involve all four limbs, the mind remaining clear. Injury at birth affecting the upper part of the cord causes **infantile spastic paralysis** (§ 596, XII.); though usually paraplegia only.

Cervical pachymeningitis, amyotrophic lateral sclerosis, and progressive muscular atrophy may produce a like effect.

Disseminated sclerosis, tabes, and other degenerative spinal lesions may terminate in generalised paralysis.

§ 612. **Landry's Paralysis** is a rare condition of ascending flaccid paralysis, of which the pathology is obscure.

The *Symptoms* start with flaccid paralysis of the legs, rapidly extending to the trunk, arms, and neck, and the muscles supplied by cranial nerves in some cases. It usually terminates fatally from involvement of the respiratory muscles, in two days to two weeks. There are no (or very slight) sensory changes, no muscular atrophy, no trophic or electrical changes, and no loss of sphincter control. The spleen is sometimes enlarged.

Etiology.—It occurs chiefly in males between twenty and thirty. It bears considerable resemblance to a rapid peripheral neuritis, and it has been suggested that it is due to an acute toxæmia of the entire lower motor neuron. Various organisms have been found in the spinal fluid by lumbar puncture, but the specific virus is believed to be an ultra-microscopic organism.

Acute diffuse myelitis may rapidly ascend the spinal cord, and cause an acute generalised paralysis. There is anæsthesia and sphincter control is lost (§ 595).

What used to be called acute spinal paralysis of adults is really an acute poliomyelitis similar to that affecting children (§ 638). Wasting is rapid, with R D, and recovery is never complete. A subacute form is also described.

General paralysis associated with extreme wasting of the muscles is seen in the later stages of the **idiopathic muscular atrophies**, and the peroneal type of myopathy (§ 640). The gradual onset in youth, the progressive course of the disease, and the hereditary history aid the diagnosis.

β. INCO-ORDINATION AND DISORDERED GAIT.

* **§ 613.** The **Gait** of all patients suffering from motor defect should be carefully studied; it will often teach us a great deal about the malady. The defect may be due to muscular weakness, to muscular rigidity, to clonic spasm, to true inco-ordination, or to want of balancing power. The most characteristic disorder of the walk occurs in the *inco-ordination* of tabes dorsalis and the sinuous or *reeling gait* due to want of balancing power in cerebellar lesions. The anatomical remarks on this subject (§ 534) may help to explain what follows.

The various diseases in which disordered gait occurs may be grouped as follows:

(a) INCO-ORDINATION (or ataxy) is met with (1) whenever the protoneurons supplying the muscles and joints are blocked as may happen in locomotor ataxy, disease of the posterior roots (*e.g.*, meningitis), and peripheral neuritis (*e.g.*, diphtheritic neuritis). Any disease affecting the posterior columns or ascending cerebellar tracts may cause it—namely, (2) *spinal tumour ;* (3) *chronic posterior spinal pachymeningitis ;* (4) *Friedreich's hereditary ataxy ;* (5) *ataxic paraplegia.* Ataxic gait is most characteristically seen in (6) *tabes dorsalis.* The joints are lax, the limbs limp and move like those of a marionette.

(b) REELING GAIT, or swaying like a drunken man, is met with most typically in (7) *cerebellar disease.* It also occurs in patients who are the subjects of (8) frequent or continuous *vertigo,* either from circulatory or other causes.

(c) The SPASTIC GAIT is a stiff gait, owing to the stiffness of the legs, due to lateral sclerosis. By degrees the patient takes to walking on tiptoe, the toes turn in, and are scraped along the ground. Later still we get " cross-legged " progression owing to the predominance in the contracture of the adductor muscles. It occurs on both sides in (9) the *spastic paraplegias* and is most frequently observed in the spastic paraplegia of children (Little's Disease), and on one side in (10) organic *hemiplegia* with descending

sclerosis; in the latter the spasticity causes the paralysed limb to be circumducted as it is brought forward in walking.

(d) FESTINATION is the gait in which the patient bends forward as he walks faster •and faster, tending to fall forward, his eyes looking fixedly in front of him. It is met with in (11) *paralysis agitans*, and to some extent in old age. In "retropulsion" the same thing happens in the opposite direction on walking backwards.

(e) In the HIGH STEPPING GAIT the patient raises his knees to an exaggerated degree. It is met with typically in (12) *peripheral neuritis* and other lower neuron paralyses attended by *foot-drop*. It also occurs in (13) *pseudo-hypertrophic paralysis*.

(f) There is a peculiar "JAUNTY" or dancing walk in (14) *chorea*, associated with excessive arm movement, which is very characteristic. Other tremors (§ 626) may render the walk peculiar.

(g) A very characteristic ATTITUDE and gait, with the head and arms hanging forward owing to the weakness of the neck muscles, occurs in (15) *post-diphtheritic paralysis* of childhood. It can be recognised as the child walks into the out-patient room.

(h) The WADDLING gait is met with in (16) *congenital hip dislocation*, advanced rickets, achondroplasia, in *coxa vara*, and all conditions of *dwarfism*. Coxa vara is a peculiar congenital condition, in which the neck of the femur forms a less obtuse angle than is usual, causing the patient to sway from side to side as he walks.

(i) The LIMPING GAIT is met with as a result of (17) *infantile paralysis* and in any (18) injury or *joint affection* confined to one side.

§ 614. Tabes Dorsalis (Locomotor Ataxy) may be defined clinically as a chronic syphilitic disease characterised by (a) disturbances of sensation, general and special, in the form of anæsthesias and lightning pains, (b) by loss of the deep reflexes, (c) by inco-ordination, and (d) by loss of the light reflex. Muscular power usually remains intact until near the end. Ten years is the average date of onset after the syphilitic infection; it rarely begins within four years after infection.

The main neurons affected are the protoneurons, notably of the spinal cord. Here the disease begins in the neighbourhood of the posterior root ganglia: the intra-spinal neuraxon (in the posterior columns) and the peripheral neuraxon (sensory nerve fibre) being secondarily affected. The distant portion of the neuraxon (i.e., that furthest removed from the neuron-body in the root ganglia) appears to be earliest affected. This is well shown in the case of the muscle-spindles. These structures have been found by physiologists for half a century in all the voluntary muscles of the body excepting the diaphragm, the intrinsic muscles of the tongue, and (?) those of the eye. Each consists of a laminated sheath of white fibrous tissue, containing a lymph space, in which lie striated fibres resembling muscle fibres, but only one-third of their diameter. Nerve fibres in great profusion enter the spindle, and Professor W. S. Sherrington has shown not only that these nerve fibres are connected with the posterior roots, but that they convey afferent impulses; and there is no doubt that these are the end organs of the muscle afferent nerves. The muscle spindles are diseased in certain cases of tabes, the intra-fusal nerve endings and contiguous muscle fibres undergoing degeneration; these changes are absent in primitive myopathies, progressive muscular atrophy, and infantile paralysis. Whenever the nutrition of a neuron is impaired, the peripheral endings of its processes are the first to undergo degeneration. Applying this principle to the pathology of tabes in the light of Sherrington's and Batten's researches, the condition of affairs may be depicted as in Fig. 166. Degeneration is also found in the end organs of other sensory nerve fibres, e.g., of the skin, joints, tendons, viscera, and nerves of special nerves (e.g., optic, acoustic). In some cases of tabes no sclerosis of the spinal cord is found. We may suppose that in such cases death has occurred before the degeneration has spread to this position.

The *Symptoms* of tabes run a prolonged course—of ten, twenty, or even thirty years. They may be divided into three stages—pre-ataxic, ataxic, and paralytic. It is of the greatest importance to recognise the malady in its earliest phase, because at this time treatment is most efficacious, and the disease may in some cases be arrested. The symptoms are most varied; loss of the knee-jerk is present in over 80 per cent. of the cases. The ankle-jerk is lost even before the knee-jerk.

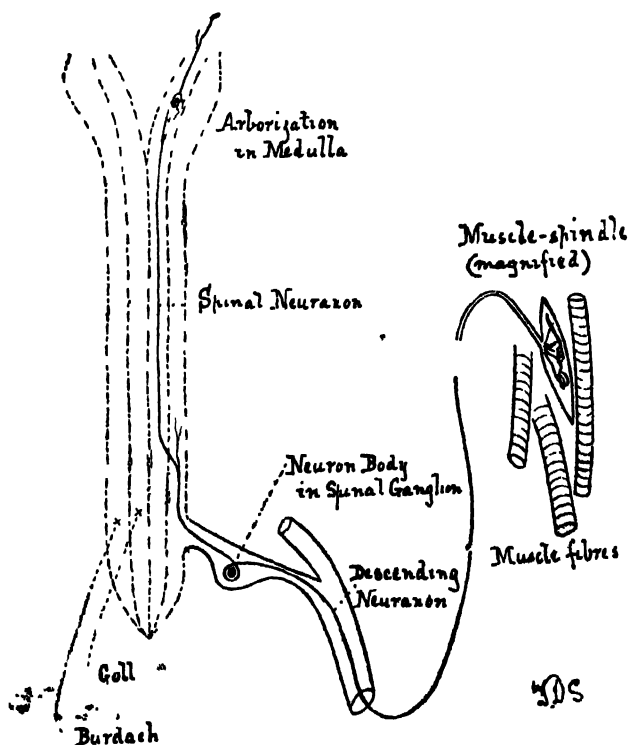(*a*) In the *Pre-ataxic Stage* the most important and characteristic

FIG 166 — Diagram of the spinal cord and muscle-sense neuron to show the PATHOLOGY OF TABES DORSALIS —The site of initial degeneration is probably in the region of the spinal ganglion.

symptoms consist of "lightning pains," vague disturbances of sensation, changes in the pupil, and other ocular symptoms. Any of these, combined with loss of knee-jerk, is almost sufficient for a diagnosis. (1) The "lightning pains" and crises of tabes are characterised by being erratic, evanescent, recurrent, and sometimes periodic. Tabes should be suspected in any neuralgias having these characters, especially of the sciatic nerves, but any sensory nerve may be affected. A "girdle-pain" is another frequent symptom in the early stages—it occurred six years before any other symptom in a case recently seen by me. Pains also occur in the situations of the various viscera, and come on in attacks, which the French writers

have named " crises." Thus, there may be attacks of gastric pain, followed, perhaps, by vomiting (gastric crisis) ; or rectal pain, which may be attended by constipation (rectal crisis) ; and so on. Frequently the bladder is the organ affected, sometimes with pain (vesical crisis), more often with a hesitation in passing water, or with actual retention, so that the patient may think that there is a stricture or enlarged prostate. Thus cases of tabes frequently come before the surgeon. Laryngeal crises are occasionally observed. They consist of spasmodic attacks of laryngeal dyspnœa, often coming on at night ; these may be mistaken for asthmatic attacks, and are very alarming, but the patient recovers in an hour or so. (2) Numerous and remarkable are the many different anæsthetic and paræsthetic symptoms observed in tabes. One of the commonest consists of scattered patches of anæsthesia, especially along the ulnar borders of the hands, and on the soles of the feet, giving rise to a sensation of " treading on cotton-wool." Sometimes there is analgesia without anæsthesia of the whole or part of the body. There is loss of deep sensation (pain on pressure) in the calves of the legs ; the joint-sense (which plays a part in the sense of posture though not so prominent a part as muscular sensibility) may be absent. Sometimes there is thermal anæsthesia ; sometimes delayed sensation ; sometimes misplaced sensation (allocheiria) ; but the strangest of all, perhaps, is what Duchenne described as the " masque tabetique," in which the patient has anæsthesia of the nose, lips, or face, which gives him the impression that he has no face. Perversions or abolitions of taste or smell are not uncommon, and diminution, heralded it may be by temporary increase, of the sexual power. (3) The ocular symptoms are very important ; the most frequent of these is transient diplopia, due to a transient weakness of one or other of the ocular muscles. Quite half the cases of tabes at one time or another suffer from diplopia. The pupils present characteristic signs. The most typical of these is the Argyll-Robertson pupil ; i.e., the pupils act to accommodation (the student will note the two a's), but not to light. Another very frequent symptom is inequality of the pupils, or they may both be very contracted or both immobile to all forms of stimulus. Irregularity of the pupils may also be present. A less frequent manifestation is primary optic atrophy, coming on with a greyish appearance of the optic disc, and terminating in white discs with clearly-defined margins. In these cases ataxia is generally absent. Any of these symptoms may precede the ataxy by ten or more years. If optic atrophy is an early symptom, the ataxy is very late in developing ; it may never develop at all. (4) The absence of the ankle-and the knee-jerk is among the earliest symptoms. (5) Various trophic disturbances are now known to be associated with tabes, thanks chiefly to the observations of Charcot. It was he who pointed out that a particular kind of osteo-arthritis, now called " Charcot's joint disease," may be present in this malady, and it may occur quite early, at a time when other symptoms are in abeyance. The joint becomes swollen, but usually it is pale, and free from pain. Indeed, there is such a complete absence of

pain, heat, and redness- -the three classical symptoms of inflammation—
that the joint (the hip, for instance) may become quite disorganised and
even dislocated ere the patient pays much heed to it.[1] In the knee-joint,
which is the favourite situation, the swelling is less likely to be overlooked.
The occurrence of such an arthritis, combined with the pupillary changes
and absent knee-jerk, is alone sufficient to establish the diagnosis. The
nails may be affected with a sort of painless ulceration ; the teeth may
be similarly affected and drop out ; or perforating ulcers of the foot may
occur. All these lesions have the same slow chronic characters.

(b) In the Ataxic Stage the above symptoms, gradually emphasised in
the course of years, are combined with (1) the characteristic walk ; and
(2) loss of equilibrium on standing. The normal power of the muscles
remains, and their nutrition is often remarkably good, but the patient
cannot control them. The typical ataxic gait has been described, and is
easily recognisable when once seen (§ 613). These patients find a difficulty
in starting to walk, but, once started, they may improve as they go on, up
to a certain point. They may also have great difficulty in turning round,
and the late Dr. Hilton Fagge has recorded an amusing incident of a patient
who once explained his unpunctual arrival at the hospital by his having
started in the wrong direction, and being compelled to continue until he
met some one who could help him to turn round and start the other way.
The arms are much less affected than the legs—at any rate, until quite
late. The patient can write or pick up a pin, but there is often a difficulty
in touching the nose when the eyes are closed. The loss of equilibrium
on attempting to stand with the eyes shut and the heels and toes together
is known as Romberg's sign, and is very characteristic of this disease
(see § 534).

(c) The Terminal Paralytic Stage is really the stage of complications,
and it may be many years before this stage sets in. The mind remains
clear in most cases until quite the end, and the patient may be able to
conduct his business for ten, twenty years or more after his locomotion has
become imperfect. By degrees the inco-ordination becomes so great that
he is no longer able to get about. Bladder complications are frequent,
and various other visceral conditions supervene ; death results from these
or bedsores, rather than from the disease itself. Perhaps the most frequent
cause of death is pneumonia. Bulbar paralysis may supervene, but the
of the most frequent of the nerve complications of tabes is general paralysis
of the insane (paralytic dementia) ; and in visiting an asylum, it is remark-
able to learn what a large number of general paralytics have commenced as
cases of tabes. Many varieties and transitional forms of these two diseases
are met with among out-patients, and may give rise to much difficulty
in diagnosis unless this frequent association is kept in mind.

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[1] In an interesting case narrated by Charcot, the patient who was doing his military
service, casually found that he could not march as well as the others, and on examina-
tion it was ascertained that the two hips were actually dislocated (" Nouvelle Icono-
graphie de la Salpêtrière," tome v.).

Causes. --(1) *Age.* Like general paralysis of the insane, tabes is almost entirely confined to adults between the ages of twenty-five and forty, and very rarely the disease occurs as the result of congenital syphilis; (2) it 'greatly predominates in the male sex—certainly over 90 per cent.[1] (3) Syphilis, often of a mild type, is the essential causal factor. The spirochætes have been detected in the spinal cords of tabetics. Syphilis affects the nervous system in two ways: the *interstitial* variety affects the meninges (*e.q.*, gummata), blood-vessels (*e.g.*, endarteritis), and neuroglia; the *parenchymatous* variety affects the nerve elements primarily. To the latter belong tabes dorsalis and general paralysis of the insane. The parenchymatous variety develops 12 to 14 years after the date of infection, and the history is usually that of a mild type of syphilis with few or no skin lesions. (4) Prolonged bodily fatigue, especially if combined with exposure to cold and malnutrition, acts as a predisposing cause. The disease is not rare in postmen, commercial travellers, and others who lead an active life.

The *Diagnosis* of tabes in typical cases- and tabes is one of the few diseases of the nervous system which usually conform to a type is not difficult excepting in the very earliest (pre-ataxic) stage. The age, sex, and syphilitic history are very significant, and the only disease apt to be confused with it at this time is general paralysis of the insane, which, however, usually presents the mental peculiarities and muscular tremor that are wanting in tabes. Compound cases occur. In the ataxic stage it may have to be differentiated from lumbar tumour, peripheral neuritis, pachymeningitis, ataxic paraplegia, and other diseases which are mentioned below. In a large majority of the cases, the Wassermann test is positive both in the blood and cerebro-spinal fluid, but in stationary cases it may be negative (§ 667). The cerebro-spinal fluid shows a considerable increase of lymphocytes and an excess of globulin.

The *Prognosis* of tabes has hitherto been regarded as hopeless —at any rate, for cure—though the disease occasionally lasts throughout the patient's lifetime without materially shortening it. But by degrees we have learned, by careful attention to clinical detail, to recognise it in its earlier stages, and much may certainly be done at that time to arrest or delay the degenerative process. The prognosis rests chiefly on three considerations—first, upon the rapidity of development of the symptoms; secondly, upon the causes in operation, especially when some of them are removable; and thirdly (the most important point), the stage at which the disease is recognised.

Treatment.—(a) For the curative *treatment.* The earlier treatment is begun the better: destroyed neurons cannot regenerate. The most

[1] The fact that tabes predominates so markedly in the male sex is interesting in connection with the theory of "forced functioning," which may be briefly stated, thus: Prolonged forced functioning of any nerve structure (especially if combined with malnutrition) results, in course of time, in atrophy and overpowering of this structure by the surrounding tissues, and its consequent degeneration. Men use their co-ordinating and muscle-sense apparatus much more than women.

promising method of treatment is the intrathecal injections of salvar-
sanised serum coupled with vigorous mercurial treatment either by
intra-muscular injections, or inunction. Twenty-four hours after a dose
of salvarsan the patient is bled, and the serum, separated from the cor-
puscles, is injected into his spinal sac after withdrawal of a small quantity
of cerebro-spinal fluid. Perfect rest in bed should be enjoined, the patient
not being allowed even to stand for an instant. The dietary should contain
a large proportion of fatty foods ; tonics may be administered to promote
assimilation, especially cod-liver oil, as being, in my opinion, the remedy
which is the best " nerve food." The system of exercises elaborated by
Dr. Fraenkel has for its object the education of the lost power of muscular
co-ordination. Maloney's exercises yield even better results. The bladder
should be emptied every four hours, as it is important to prevent paralysis
of that viscus. Patients should certainly avoid anything like fatigue of
mind or body. Sexual excess, exposure to cold, should be avoided, as
well as alcohol and tobacco, except in great moderation. (*b*) In regard to
Symptomatic Treatment, the most important symptoms calling for treat-
ment are the lightning pains. Morphia must be forbidden, for with re-
current pains like these the habit is apt to be developed. On the other
hand, the same treatment which cures or modifies the disease also relieves
the pains. Phenacetin, antipyrine, pyramidon, hyoscyamus, belladonna,
physostigmine, Indian hemp, and other analgesics, combined with rest
and warm baths, are often successful.

Whenever the **posterior columns of the spinal cord are affected,** whether primarily
or secondarily, an ataxic gait and other symptoms resembling tabes may arise. Severe
anæmias and other toxic debilitating conditions may be complicated, as Dr. James
Taylor and others have shown, by posterior sclerosis and ataxic gait. There are four
named DISEASES OF THE SPINAL CORD, all of which, compared with tabes dorsalis, are
relatively rare, but give rise to inco-ordination of the gait resembling tabes—viz., spinal
pachymeningitis, spinal tumour involving the posterior columns, Friedreich's disease,
and ataxic paraplegia.

§ 615. **Spinal Meningitis** is apt sometimes to be confined to, or at any rate to
predominate in, the posterior region of the theca vertebralis involving the dorsal
spinal roots, and in some cases I have seen has given rise to disordered gait and other
symptoms characteristic of tabes dorsalis. I believe that many cases of so called
tabes cured by iodide have been due to syphilitic meningitis involving the dorsal roots
(*op.* § 596). One is aided in recognising this condition by the long history of continuous
pain and tenderness in the spinal column, and continuous pain shooting down the
spinal nerves. These considerations have led me to describe as a separate condition—

Posterior Spinal Meningitis.—It is extremely chronic, and not infrequent. The
symptoms are few and ill-marked, on which account such cases rarely find their
way into hospitals. They get into the infirmaries, however, and my experience at
the Paddington Infirmary led me to the conclusion that the disease is fairly common.
At a meeting of the Neurological Society of London held at the Infirmary in 1890, I
was able to show the cords of five fatal cases and three examples of the affection during
life. Here is a fairly typical case : A woman, æt. seventy-two, was admitted into the
infirmary in January, 1890, with all classical symptoms of tabes preceded and accom-
panied by very severe and lightning pains. The history, briefly, was that for three
years prior to admission she had suffered from paroxysmal pain shooting down the legs
and various other places, and that she had had other attacks which resembled gastric

crises. For two years prior to admission she had suffered from ataxy which became progressively worse, and at last so marked that she was unable to stand. Many careful observers saw the case; all had no doubt it was an example of locomotor ataxy occurring in a woman. She died of pneumonia in June, 1891, and at the autopsy, though the pia mater and arachnoid on the ventral surface of the cord was normal, that on the dorsal surface was thickened and opaque. This thickening was irregularly distributed from end to end, and was most marked in certain patches of 3 inches in length in the mid-dorsal and in the lumbar regions, in which positions it was about the thickness of a piece of wash-leather, and showed commencing calcareous plates. These patches were firmly adherent to the posterior aspect of the cord, and the posterior columns beneath were sclerosed as in cases of advanced tabes.

Spinal Tumour (intra- or extra-rachidian) pressing on the back of the cord, especially in the *lumbar region*, may be attended by ataxic symptoms closely resembling those of tabes.

§ 616. Friedreich's Disease (Hereditary Ataxia) is a rare hereditary condition occurring in children, often sisters and brothers of the same family. There are five differential characters. (i.) The ataxy is often most marked in the arms. There is inability to stand with the eyes shut, often loss of the patellar reflex. On the other hand, Babinski's sign is generally present. There are no lightning pains, no crises, and no Argyll-Robertson pupil. (ii.) Tremors of an ataxic nature come on later in the arms, so that the hand in approaching the mouth does not reach its goal, its movement being accompanied by jerky, irregular movements of the head and neck. (iii.) The speech is impaired in the same manner as in disseminated sclerosis. (iv.) Nystagmus is present in most cases. (v.) There is usually no mental change. The muscles become weakened, and deformities such as scoliosis and talipes ensue, notably pes cavus.

The degeneration involves the posterior columns, the pyramidal tracts, Clarke's columns and the direct cerebellar tracts.

Ataxic Paraplegia is another rare affection. It is due to sclerosis affecting the posterior as well as the lateral columns, and it results in symptoms of the two lesions. It is generally an early stage of disseminated sclerosis. From tabes it is differentiated by the spastic rigidity, increased knee-jerks, and ankle-clonus. The Argyll-Robertson pupil and lightning pains are absent.

§ 617. In Cerebellar Lesions.—Cerebellar inco-ordination. Peripheral proprioceptive impulses to the cerebrum not being blocked in cerebellar disease, both the vestibular sense and the musculo-arthritic sense remain intact. A patient with cerebellar inco-ordination is thus able to feel the position of his head and his limbs. Hence it is that the direction of his movements is fairly accurate, and that the inco-ordination is but little affected by the opening and closing of the eyes.

Cerebellar *inco-ordination* manifests itself in the following ways :

(a) The movements are characterised by *intention tremor*, i.e., the tremor is absent during repose and occurs during the execution of a movement. One of the functions of the cerebellum appears to be the fusing of individual contractions into one steady continuous contraction.

(b) Muscular contractions are not properly graded as to strength—*dysmetria*; they tend to occur suddenly and in an exaggerated degree. Thus in attempting to touch the tip of the nose the finger shoots abruptly beyond the goal.

(c) The movements of individual skeletal segments which go to make up a complete movement are not properly timed —*asynergy*. Thus in bringing the heel to the buttock, the patient instead of simultaneously

flexing the thigh and the leg, first flexes the thigh with the leg extended, and then flexes the leg. Again in walking the trunk lags as it were behind, and it may be necessary for the observer to push it forwards in order to preserve the balance.

(d) Each phase in a complex movement requires to be inhibited before the succeeding phase can be effected (diadoco-kinesis). In cerebellar inco-ordination the intervals between the individual components of such a movement tend to be prolonged—*adiadoco-kinesis*. Scanning speech is an instance of this defect, also the inability to pronate and supinate the forearm in rapid succession. This is not absolutely pathognomonic of cerebellar disease.

(e) When asynergy is pronounced a *cataleptoid* tendency may be displayed when a limb, especially the leg, is extended : when the patient is told to raise both his legs from the bed, he is able to keep them raised in a state of absolute fixity for a much longer time than is possible for a normal person.

Cerebellar hypotonus and *myasthenia.*—Hypotonus is often mentioned as a feature of cerebellar disease. In unilateral disease there may be slight hypotonus on the corresponding side. The myasthenia is probably caused by the effort of voluntary co-ordination.

Cerebellar tumour.—This is the most frequent of all the intracranial tumours. The symptoms are : vertigo, optic neuritis, headache, vomiting, inco-ordination (manifesting itself by intention tremors, nystagmus, dysmetria, asynergy, adiadoco-kinesis (e.g., slow-speech), defects of equilibration during walking rather than in standing), hypotonus, and myasthenia.

Of these vertigo is the most constant. Optic neuritis occurs in 80 per cent. of the cases, and in unilateral tumours generally begins, and is most marked, on the side of the tumour. The headache in many of the cases is referred chiefly to the occiput and back of the neck. The nystagmus is generally more marked and slower when the eyes are turned towards the side when turned in the opposite direction. In walking the patient feel towards the side of the tumour, and in the rare cases of actual rotation he rotates from the supine to the prone position towards the side of the tumour. The tendency to look and to lurch one side may cause the patient, by way of compensation, to turn the body and the head to the opposite side, and this may deceive the observer. The difficulty in equilibration is kinetic, not static : the patient may be able to stand still even with closed eyes.

In unilateral tumour hypotonus and inco-ordination (tremor, dysmetria, asynergy, etc.) are most marked on the side of the tumour.

Tumour of the middle lobe may cause a tendency to fall forwards or backwards. Rarely head retraction and opisthotonus occur.

The vertigo, vomiting, optic neuritis, and headache are due to pressure on surrounding structures. Pressure on the brain-stem may cause augmented deep reflexes and descending lateral sclerosis ; pressure on the

fourth ventricle, hydrocephalus. Pressure on the cranial nerves may give rise to a variety of cranial nerve palsies.

Abscess of the cerebellum.—The symptoms are very similar to those of cerebellar tumour.

Hæmorrhage.— This is rare, and generally leads to rapid coma and death. Defective equilibration is a prominent feature.

Embolism and *thrombosis.*—The cerebellum derives its blood supply from the vertebrals. There is little tendency for emboli to travel up these vessels. Moreover, the free anastomosis between the cerebellar arteries renders widespread softening unlikely. The symptoms are similar to those of hæmorrhage.

Congenital defects of the cerebellum.—There may be complete absence of the cerebellum : more commonly one hemisphere is wanting ; or the organ may be small or ill-developed. Motor disturbances of the cerebellum type are present.

Atrophy of the cerebellum. —Gordon Holmes makes four principal types of cerebellar atrophy of which one is acute. (1) Primary parenchymatous degeneration, a familiar affection. (2) Olivo-ponto-cerebellar atrophy, a chronic degeneration coming on in advanced life ; the central nuclei remain intact. (3) Progressive degeneration from vascular and interstitial disease. (4) Acute non-suppurative encephalitis occurring in children after zymotic disease. The child may become unconscious and suffer from convulsions ; during convalescence it is found to be ataxic in all four limbs, and there is often articulatory defect. Recovery takes place in a period varying from a few weeks to a few years. Symptoms akin to those occurring in these four classes may occur in (5) Degeneration of the spino-cerebellar tracts, and (6) Congenital smallness of the central nervous system with cerebellar symptoms.

(γ) Increased Muscular Action.

Involuntary increased muscular action may be—
Continuous, when it is known as rigidity or   § 618
Intermittent, when it is known as *tremor* if the movements are small and vibratile, and *clonic spasm* if the movements are large   § 626
attacks of violent muscular movements, often associated with disturbance of consciousness, are known as *convulsions*   .. § 634
the patient presents a continuous stiffness or rigidity in the affected muscles
— Spasm.

Tonic Muscular Spasm or rigidity is indicative of an irritative lesion functional or organic, in the motor tract. It is mainly due to withdrawal of the normal inhibitory influence.

> I. Paralytic rigidity (early and late).
> II. Hysterical rigidity.
> III. Occupation cramp.
> IV. Cramp.
> V. Tetanus.

VI. Tetany.
VII. Hydrophobia.
VIII. Thomsen's disease.
IX. Arthritic rigidity.
X. Progressive lenticular degeneration.
XI. Paralysis agitans.

The first and tenth of these causes are of organic origin ; the second, third, and fourth are functional ; the fifth, sixth, and seventh are toxic ; the eighth is due probably to some hereditary defect ; and the ninth is probably reflex.

§ 619. Paralytic Rigidities of Organic Origin are associated with paralysis, and are of three kinds, as exemplified in hemiplegia.

1. *Early Rigidity* is that which comes on in the paralysed muscles in cases of hemiplegia due to hæmorrhage within the first few days. It is due to pressure of the clot, or to an area of congestion surrounding the clot. It generally passes off within a few days.

2. *Late or Spastic Rigidity* of the muscles is that which comes on gradually in the course of a month, sometimes as early as a fortnight in all cases of paralysis due to lesions of the upper motor neuron. It is met with typically in hemiplegia, spastic paraplegia, and all lesions followed by *descending lateral sclerosis* in the spinal cord. It is associated with increased knee-jerks, ankle-clonus, and Babinski's sign ; tremors or clonic muscular spasms or athetosis may also be present. It comes on gradually, and increases progressively. It diminishes during sleep. Warmth lessens it, also chloral hydrate.

3. *Organic Contracture* ensues in all paralysed muscles in the course of years, whether of the upper or lower neuron type (such as hemiplegia and infantile paralysis respectively). It is due to an atrophic fibrosis of the substance of the paralysed muscles.

4. *Other organic rigidities.* Meningitis and other lesions at the base of the brain, notably in the region of the cerebellum, may give rise to rigidity of the cervical muscles, causing retraction of the head. Acute spinal meningitis is apt to cause rigidity of the spinal muscles. Painful joint affections may lead to rigidity of peri-arthritic muscles to secure rest ; similarly a protective rigidity of the abdominal muscles result from disorders of the abdominal viscera (*e.g.*, peritonitis—).

Hysterical Rigidity generally comes on quite suddenly, after an emotional storm. It is often of limited extent, involving only one foot (hysterical club-foot), and it may give rise to attitudes and deformities. It is often accompanied by local and loss of cutaneous reflex. It may pass off as suddenly. It may last for years. This rigidity tends to persist during patient, generally a female, presents other signs of hysteria.

§ 620. Occupation Cramp and other **Occupation Neuroses.**— already seen that exhaustion produces muscle-cramp. The continued use of a certain group of muscles is apt to produce five symptoms, localised to that group, but tending ultimately to spread to other

if the cause continue in operation. In order of frequency, they are (1) tonic spasm, (2) paresis, (3) pain, (4) tremor, and more rarely (5) atrophy or hypertrophy.

Writer's Cramp is the most frequent example, and may be taken as a type, but what follows applies almost equally to other occupation neuroses. (i.) *Tonic Spasm.*—In scrivener's palsy, after writing for some time, the fingers get so stiff that the patient cannot write. Sometimes the spasm is very painful, and occasionally there are twitchings. The character of the writing alters, and in the course of weeks or months the slightest attempt at writing produces a tonic spasm in the muscles used for that purpose. For a long time *tonic spasm appears only on attempting to write*, the hand in the intervals being quite free from symptoms, but in the later stages the spasm may become persistent or start spontaneously. In exceptionally severe cases it may even spread from the hand to the arm, shoulder, neck, and other parts. In the early stage the general uses of the limb are unimpaired ; a patient affected with writer's cramp may paint, or play the flute, but in most cases other delicate manipulative procedures cannot be performed with the same precision as formerly. (ii.) The *Power* of the grasp as tested by the dynamometer is said to be normal, but this is not a delicate test of the small muscles involved in writing. By careful examination I have very rarely failed to detect some loss of power. Poore also insists that definite slight weakness of certain muscles of the hand is not uncommon in writer's cramp. (iii.) Some discomfort is always experienced and this sometimes amounts to actual pain. This pain, moreover, has a great tendency to spread. In some cases the pain is the most pronounced feature of the case, and is accompanied by tenderness of the nerve-trunks, and tender points elsewhere (occupation neuralgia). " Pins and needles " and other subjective symptoms are by no means uncommon but I am not aware that anæsthesia is ever observed. (iv.) Atrophy is said to be rare, but a careful comparison of the interossei and other muscles of both hands in several cases has convinced me that it does sometimes occur. Hypertrophy is much more frequent ; it usually follows those in which spasm is the leading feature. (v.) Electric irritability is increased in the earlier stages, and slightly diminished in the later to both faradism and galvanism, both in the muscles and the nerves. Twitchings and clonic spasms are only occasionally observed tremor and unsteadiness are uncommon.

Prognosis.—The advent is very gradual, and the effect may last years if the cause is not removed. The prognosis depends on the duration of the affection and the stage when the treatment is commenced ; (ii.) the ability of the patient to cease the occupation ; and (iii.) intelligence in grasping the principles of treatment.

Diagnosis, by reason of its association with the causal occupation, is usually difficult, but there are several organic and functional disorders (brachial neuritis (*q.v.*), lead palsy, and even disseminated sclerosis) may be mistaken for writer's cramp at their outset ; but in such

careful inquiry shows that even from the first the symptoms were not solely determined by the act of writing.

Treatment.—The disease is most frequent, in nervous, sensitive persons, especially when suffering from a general " lowered tone," or malnutrition, anxiety, or grief. The fact that there is but one determining cause should render the treatment comparatively simple, but, as a matter of fact, it is very difficult because the malady is chiefly found in those whose daily bread depends upon the performance of a certain muscular movement. Careful re-education can, however, accomplish a great deal, and particularly the adoption of a freer and larger style of writing, *by holding the pen more loosely and in a new way.* The patient should learn to hold the pen between the first and second fingers with the *back* of the hand against the paper, and practise writing a series of large sloping M's. A certain amount of rest—at any rate, at the outset—is indispensable. Meantime the patient should begin learning to write with his left hand. Sedatives are of service, especially in the neuralgic forms, such as chloral, bromide, small doses of morphia, physostigma, Indian hemp, belladonna. [...] ¹⁄₁₀₀ grain (0·0006) hypodermically two or three times a week. Faradism does harm, but galvanic electricity has often seemed to me beneficial, especially when combined with massage. Gymnastic exercises are strongly advocated by some. One remedy—cod-liver oil—has rendered great service, and some of the most successful cases I have seen have been treated by a combined method of moderating the amount and improving the style of the writing, and the administration of small doses of bromide and cod-liver oil.

Other Occupation Neuroses resembling the foregoing are found among telegraphists, drapers (in using scissors), cigarette rollers, violin-players, piano-players, typewriters—in short, among those following any occupation necessitating the constant repetition of one particular movement.

Cramp is a tonic muscular spasm occurring in one or more muscles of a limb. It is a tro[...] frequent symptom in persons apparently in good health. It is most apt [...] at night. A muscular cramp of great severity may seize [...] all the limbs, and even the respiratory and trunk muscles [...] and prove deadly unless aid is close at hand. It is apt to come on in those [...] muscles are exhausted, but a low temperature of the water plays some part in [...] ction. Some persons seem prone to be affected by cramp throughout life on slight [...] ation, such as lying with [...] a strained position. Others only suffer [...] when their digestion or [...] is out of order. The best remedy [...] of bed and gently move and rub the limb. Bromide will generally prevent [...] nce, and any lithæmia, gouty or rheumatic tendencies should be corrected. [...] a frequent premonitory symptom in peripheral neuritis and phlebitis. It [...] with in subjects of Bright's disease and gout, and cramp of the legs is a painf[...] in the first stage of cholera.

§ 621. **Tetanus** is a severe disease characterised by paroxysms of tonic [...] times clonic spasms, due to the inoculation into a scratch or wound of a specif[...] whose chief habitat is in the earth.

Symptoms.—(1) Within a few days after the injury the patient complains [...] ness of the jaw and back of the neck. (2) Very soon these muscles become [...] d. The spasm of the jaw-muscles is known as trismus, or lock-jaw. A similar tonic [...]

spreads to all the muscles of the trunk, and in a less degree of the extremities. The back is rigid, sometimes arched in the position of *opisthotonos*, in which only the head and buttocks rest on the bed. Or there may be flexion to one side—*pleurosthotonos*, or bending forward of the body—*emprosthotonos*. The angles of the mouth are drawn down, and the eyebrows are elevated—*risus sardonicus*. (3) Clonic spasms supervene from time to time, in which the already rigid muscles become still more contracted, causing agonising pain. The slightest touch may excite clonic spasms. In severe cases these spasms become more frequent, leading to death from involvement of the glottis or respiratory muscles. (4) The temperature may be normal or slightly raised throughout, and may rise to 108° F. just before death. There is often retention of urine. The mind is clear to the last. A local form due to head wounds is described, with paralysis of the facial muscles and difficulty in swallowing.

After an injection of anti-tetanic serum to obviate the disease, *localised spasm* may result, *e.g.*, in one arm in the case of wound in the hand.

Diagnosis.- -In hydrophobia the spasm is at first clonic. It affects chiefly the muscles of respiration and deglutition, rendering it difficult to swallow even water, and there is more mental agitation than in tetanus. In strychnine poisoning the muscles relax in the intervals between the spasms, and the spasms involve the extremities to a greater degree. In spinal meningitis there is a temperature, and there is no trismus. Tetany is not likely to be mistaken for tetanus. In hysterical opisthotonos there are other evidences of hysteria. Trismus is caused also by disease of the pons and acute bulbar paralysis, and in association with periostitis of the jaw, disease of the temporo-maxillary joint, or other local irritation, such as the cutting of a tooth; but the course of the disease serves to differentiate these from tetanus.

Prognosis.—Death occurs in acute cases in one to twelve days from exhaustion or involvement of the glottis or respiratory muscles. In more chronic cases the spasms become less frequent, and recovery ensues in two or more months. If the disease does not at till more than twelve days after the injury, the outlook is not so hopeless. The mortality in untreated cases is very high—about 90 per cent.

Etiology.- -Tetanus is caused by a wound, however trivial, into which the tetanus bacillus has entered. Tetanus of the new-born is due to want of aseptic precautions in treating the navel. The bacillus has its habitat in the earth. The symptoms are caused by *the actual union of the toxin* with the affected neurons within the central nervous system, the toxin travelling up the nerves from the seat of infection.

Local *Treatment of the* wound is necessary by excision, cautery or antisepsis. For the spasms, chloroform, bromides, chloral hydrate, and other drugs are given. The tetanus antitoxin has given good results in the European war. It is valuable both in obviating an anticipated attack and in curing an active attack, the great thing is to give it promptly. The intra-thecal method (by lumbar puncture, § 666) is the best; the intra-venous method nearly as good. Little benefit results from subcutaneous or intra-muscular injection. The intra-cranial method is only resorted to in desperate cases. The anti-toxin unites with the toxin, rendering it harmless.

§ ___. ___ a muscular stiffness occurring in spasms, affecting mainly the ends of the extremities.

It comes on mostly in infancy in the ___ paroxysmal stiffness affecting ___ forearms, hands, and feet. The attitude of the fingers compressed in ___ (the accoucheur's hand) has been emphasised, but it is unessential; it is ___ assumed in athetosis and many other tonic spasms. The paroxysms ___ few seconds to an hour or so, and in severe cases there is no intervening ___ In severe cases, moreover, all the muscles of the body are affected, ___ may even be opisthotonos. There is a good deal of neuro-muscular irrit-___ "spasmophilia "—to percussion and to both forms of electricity, and if the ___ drawn down the face, a wave of muscular contractility follows it (Chvostek's ___ It is often associated with laryngismus stridulus. Many grades of severity ___ the disease may only last two days or two or more months, recovery being ___ general but not invariable rule. The *Diagnosis* is not difficult. In tetanus the ___ mainly affects the jaw, and there is a history of a wound.

Etiology.— Tetany is most frequent in children the subjects of rickets or diarrhœa, or other gastro-intestinal disturbance, but it is not solely confined to childhood., In adults it may be associated with dilatation of the stomach or any other gastro-intestinal condition attended with fermentation. Cases have been noted in association with pregnancy and albuminuria. An endemic form of tany occurring chiefly in the winter months and among shoemakers is met with in certain continental towns. The production of alkalosis of the blood by forced breathing may induce tetany. Several observers have suggested that tetany might be due to an excess of sodium or potassium in the blood, seeing that the administration of these alkalies for acidosis may induce the condition, but there is no evidence that children suffer from alkalosis. Tetany invariably occurs after removal of the parathyroids. Schaefer suggests that these glands yield a secretion which inhibits muscular contraction.

The *Treatment* should be directed to the causal condition. Bromide, chloral hydrate, and immersion in cold water will relieve the spasm.

§ 623. Hydrophobia is due to inoculation by the saliva of an animal suffering from rabies, and is characterised by spasms of the muscles, notably those of deglutition and respiration.

Symptoms.—(1) After an incubation stage which is generally about six weeks, never less than twelve days, and sometimes as long as twelve to eighteen months or more, there is an insidious onset of malaise,with perhaps slight fever, and sometimes tingling in the wound. (2) With or without premonitory symptoms paroxysms of painful spasms of the pharynx supervene, coming on at first with a slight stiffness, and brought on by any attempt to swallow, even water : hence the hydrophobia. (3) These spasms, at first clonic, become tonic, lasting a quarter to half an hour at a time, and spread to the muscles of respiration and of the neck. The attacks produce excruciating pain and agony of mind. The mind is quite clear, but in the intervals there are prostration and general hyperæsthesia. (4) Paralysis ensues in three or four days' time first of the muscles of the lower jaw, and death follows within a week from the onset.

Treatment.—Suction and cauterisation of the wound immediately after the bite may be employed. The immunisation treatment of Pasteur is dealt with in § 418. Recourse may be had to narcotics, chloroform inhalation, and chloral.

§ 624. Thomsen's Disease (Congenital Myotonia) is a rare condition characterised by a spasm of the muscles when the patient attempts voluntary movements. Thus on trying to rise or walk his legs become rigid. After walking a short distance the stiffness tends to wear off. Similarly after tightly closing his hand it may be some time before he can relax the grip ; or if he shuts his eyes he may not be able to open them at once. The spasms are increased by cold and nervousness. The muscles are generally large, but not correspondingly strong. Under the microscope the fibres show hypertrophy with proliferation of their nuclei. They manifest increased excitability both to mechanical and electrical stimulation, the resulting contraction persisting for some time. K.C.C. is not greater than A.C.C. The disease is often familial. It does not appear to shorten life. Warmth and continued activity relax the spasm to some extent.

Arthritic rigidity is known by its being associated with some joint which though the latter may be very slight. It affects both the extensors and flexors of the joint, though chiefly the flexors. I have usually noticed that it is increased dur

Progressive Lenticular Degeneration is a rare disease, first fully describe
S. A. K. Wilson in 1912. It usually attacks several members in a family,
hereditary. It affects the young of either sex, and is characterised by sp or
hypertonicity of the muscles, with contractures, weakness, and tremor on movent.
The face is set in a spastic smile ; the limbs may be contracted as in hemipleg nt
can be moved at will, as there is no true paresis. Dysphagia, dysarthria or an ria
ensue. Emotionalism and mental symptoms may occur. The reflexes are norml.
Emaciation is progressive, and death takes place in from six to five y s.
Cirrhosis of the liver, unsuspected during life, is found after death. The dis

is due to bilateral degeneration of the lenticular portion of the corpus striatum.

§ 625. **Decerebrate rigidity** may be caused by any disease which interrupts the neuraxis at the level of the superior corpora quadrigemina, such as hæmorrhage, tumour, and meningitis leading to internal hydrocephalus. Partial interruption may give rise to partial rigidity. (See § 534.) In irritative lesions the rigidity may occur in the form of tonic fits.

Rigidity is a pronounced feature of **Encephalitis Lethargica** (§ 587). The face presents a mask-like appearance and the limbs tend to maintain any position into which they are passively placed. The patient may develop a condition closely resembling paralysis agitans, showing that the virus exercises a selective action upon the corpus striatum.

A similar rigidity is met with in paralysis agitans.

There are irregular **movements** *or shaking of the affected muscles, the range of the movements being either small* (TREMOR), *or large* (CLONIC SPASM).

§ 626. **Tremors** and **Clonic Muscular Spasms** constitute a frequent and prominent symptom in many different nerve lesions. For clinical purposes abnormal muscular movements may be divided into tremors or movements of small amplitude, and clonic spasms or movements of larger range ; paralysis agitans may be regarded as a type of the tremors, chorea as a type of the clonic spasms. The movements we are now considering must not be confused with generalised convulsions or fits (§ 634), nor with the uncertain movements of paralysed limbs or tabes dorsalis.

Classification.—It will be convenient to consider first the diseases in which tremors occur (such as paralysis agitans), and later those characterised by clonic spasms (such as chorea). But this division must not be taken too absolutely, for movements which are commonly small are occasionally apt to be large, and *vice versâ*.

TREMORS.

Partial Degenerations.
- I. Paralysis agitans.
- II. Senile tremor.
- III. Disseminated sclerosis.
- IV. Progressive lenticular degeneration.

Functional.
- V. Hysterical trembling.

Toxæmic.
- VI. General paralysis of the insane.
- VII. Alcoholic tremors.
- VIII. Metallic tremor.
- IX. Other toxic tremors.

Nervous or neurasthenic tremor, and Graves' disease.

Organic.
- X. Organic diseases in which tremor is not always a prominent feature :
  - (1) Post-paralytic tremor.
  - (2) Amyotrophic paralysis.
  - (3) Lateral sclerosis, Friedreich's disease, and other degenerative conditions.

CLONIC SPASMS.

Generalised.
- I. Chorea.
- II. Hysterical spasms.
- III. Myoclonus multiplex.
- IV. Progressive lenticular degeneration.

Localised.
- V. Habit spasm.
- VI. Facial spasm.
- VII. Spasmodic torticollis.
- VIII. Other localised spasms.

Organic.
- IX. Post-paralytic clonic spasms.

CLINICAL INVESTIGATION.—The more important points to be observed about cases of tremor or clonic spasm (in addition to their range) are : 1. Their *distribution*, which may be localised to one limb, as in hysteria, or generalised, as in alcoholic tremors.

2. The *rhythm* of the tremor should be noted : for example, it is regular in paralysis agitans, senile tremor, Graves' disease, small hysterical tremors, alcoholic and metallic tremors ; whereas it is irregular in the various forms of chorea.

3. It should always be ascertained whether the tremor is present when the limb is *resting* on the bed or table or only when the muscles are in action. Some kinds of tremor come on only during muscular action, as in disseminated sclerosis. This is known as *intention* tremor. Other kinds of tremor, such as paralysis agitans, persist during muscular rest—*e.g.*, when the limb is laid on a table, and may even show a disposition to lessen on voluntary movement. It should, however, be borne in mind that most kinds of tremor tend to be increased when the muscles are thrown into action, when the patient's attention is directed to them, and during emotional states.

4. Sleep causes a cessation of the movements.

5. The *mode of onset* does not afford much valuable information It is apt to be sudden in all forms of hysterical and functional spasm, but in most of the remainder it is gradual.

6. The *age* of the patient often gives us an important clue. Thus senile tremor occurs in the aged ; paralysis agitans nearly always after middle life ; whereas alcoholic tremor, metallic tremor, and paralytic dementia affect persons in the middle decades of life ; and, finally, hysterical tremor, chorea, disseminated sclerosis, and habit spasm are met with chiefly in the comparatively young.

7. *Causation and Pathology.*—Tremors and clonic spasms may arise under one three pathological conditions : (1) Some organic lesion of the nervous system, so as disseminated sclerosis ; (2) .... or toxæmic causes, such as hysteria, ... debility, anæmia, alcoh......... ...... and (3) reflex irritation, such s a ay be seen in the case ..., when the tremor may disapear on the removal of .... tion of an error of refraction, or... in a case I have seen, after a pharyngeal affection has been remedied.

A study of the organic cases throws much light on the pathology of tremors and clonic spasm, for we find that they are always due either to partial destruction or imperfect recovery of some part of the motor tract. The greater frequency of post-hemiplegic clonic spasms (1) after embolic lesions, which are less destructive than hæmorrhage ; (2) after hemiplegia associated with hemianæsthesia, which indicates a lesion far back in the internal capsule ; and (3) after the hemiplegia of children, in whom the recuperative power is correspondingly great—all point to this conclusion. Turning to other diseases, that disseminated sclerosis is attended by an incomplete destruction of the motor tract. Paralysis agitans, again, is evidently one of the senile degenerations, which is very gradual, and therefore for a long time incomplete.

§ 627. Paralysis Agitans (Synonyms : Shaking Palsy, Parkinson's Disease).

—The onset is extremely gradual, and the three cardinal symptoms are (i.) tremor, which is moderate in amplitude, rhythmical, and of general distribution, excepting the head and neck. It generally begins hemiplegically. The fingers and thumb are approximated in a " pill-...... " movement. Dr. Purves Stewart has pointed out that the toes...... a similar tendency to curl over the sole. The tremor continues when the limbs are supported. It tends to diminish on voluntary movement. It always predominates on one side of the body, and the onset is often accompanied by " rheumatic " pains. (ii.) The muscles gradually become stiff, and the attitude and aspect of the patient (Fig. 2, § 16) are very characteristic. The head appears to be fixed, and is bent a little forward ;

and the patient walks and turns round rigidly, as if "made of glass." This rigidity may precede the tremor by many years. The gait is characteristic; the patient walks faster and faster, tending to fall forward (festination). If pushed backwards, he continues to walk backwards, unable to stop until he meets an obstacle (retropulsion). The patient's face appears like an expressionless mask with the eyes always looking forwards, with a fixed "reptilian" stare. (iii.) The patient is nearly always over forty years of age, and, in 65 per cent. of the cases, of the male sex. (iv.) The other and less important points for differential purposes are progressive weakness, which gradually involves all the limbs; the speech, which is apt to be drawling, indistinct, monotonous, and slow; and various subjective sensations, such as restlessness and a constant desire to be moved, if in bed. The intellect is preserved, and though the disease lasts for many years, it does not usually shorten life. Towards the end of life the tremor disappears, and is replaced by paralysis, rigidity, and contractions.

The pathology of paralysis agitans has not been completely settled. Some years ago Dr. Robert Maguire and the author, by examining the nerves after death, found varying degrees of degeneration in the different peripheral nerves and other parts of the nervous system, but there is no doubt that the essential lesion is one involving the corpus striatum. It seems probable that the degeneration is ████ ████aturely senile one, and is brought about by a peculiar cond████ ████

A Senile tremor is described by some, ████ ████mary experience I was rarely able satisfactorily to differen████ ████ agitans from senile tremor. However in the latter ████ tremor always involves the head and neck, being, indeed, sometimes most marked in or limited to th██ ██tion, and rigidity and paresis are less marked.

§ 628. Disseminated Sclerosis.—Islets of sclerosis in the spinal cord were described by Cruveilhier as an anatomical condition, but the clinical symptoms attaching to the condition were not recognised until Professor J. M. Charcot studied the disease clinically, and gave to it the above appropriate name. Various types of the disease are described, according to the ████ chiefly involved. The three characteristic symptoms of this disease are (i.) the tremor, which is rhythmical, rather larger than paralysis agitans, and occurs on██ ██hen the muscles are in action, ceasing when the limb is supported. This is, inde██ ██e most typical of "intention tremors." It involves all the muscles, including ████ of the head and the tongue. The intention character differentiates the tr████ ██paralysis agitans, and it may be distinguished from chorea by the fact that the ██████ goes straight to its goal, as, for instance, in feeding, whereas in chorea it ta████ ██ing course. (ii.) The patient is usually under thirty (the earliest case I have ████ was sixteen years old). It rarely commences in persons over forty-fi██ ██y years of age. The sexes are equally affected. (iii.) The speech is character-i██ ██g slow and syllabic, the patient pausing between the syllables of a word and ██ ██ing the labial consonants. There is a torpid condition of the intellect, especially in those cases where the brain is also involved. (iv.) Vertigo is an important and early symptom, occurring in something like 70 per cent. of the cases. (v.) Nystagmus is another frequent symptom, and it is often accompanied by primary optic atrophy (white atrophy). (vi.) Various other symptoms may arise, according to the position of the sclerosed patches. Thus, if the lateral columns be much involved, as they are in the majority of the cases, we get spastic symptoms with increased

deep reflexes; if the posterior columns, tabetic, and so on. In an analysis of fifty cases, Dr. F. S. Palmer found that spastic weakness, especially of one limb only, was the first symptom noticed in the disease in fifteen cases. The abdominal reflex is absent. Various sensory disturbances may also be observed. Primary optic atrophy may occur. (vii.) The course of disseminated sclerosis is prolonged; it may last from two to ten years, being longest in the purely spinal form, shortest in the cerebro-spinal form.

In **Progressive Lenticular Degeneration** the tremor occurs on voluntary movement, the muscles are in a condition of hypertonus (§ 624). Excitement and effort increase the tremor.

Hysterical Tremor is a very frequent manifestation of that diathesis. It may be small and regular or large and choreiform (see below), also "intentional."

General Paralysis of the Insane has been fully described in Mental Disorders. Here the tremor is very fine, regular, and vibratile and it tends to affect the lips and tongue more than any other parts; indeed, at first these alone may be involved. It is accompanied by more or less general weakness and by a characteristic mental alteration.

Alcoholic Tremor is one of the most constant evidences of chronic alcoholism. It is especially noticeable in the hands, is small and vibratile, and worst in the morning. There is also a history of dyspepsia with morning vomiting, insomnia, and other evidences of chronic alcoholism are present.

Metallic Tremor.—Mercury, lead, and zinc, especially when introduced into the system in the form of vapour, or in small doses for a considerable time, not infrequently produce tremor. It is small, rhythmical, and only apparent when the muscles are in action. It is accompanied by the other signs of poisoning by the respective metals. Mercurial tremors occur mainly amongst thermometer-makers and water-gilders in the manufacture of mirrors. Lead tremors occur largely amongst the Cornish and Cumberland lead-miners, and zinc tremors amongst brass-founders who are exposed to the fumes of oxide or oxychloride of zinc.

Other toxaemic conditions are also attended by tremor, such, for instance, as diabetes, malaria, influenza, trypanosomiasis, and pyrexial states. Here the tremors are small and rhythmical, and a history of the cause is easily obtainable. **Graves' disease** is attended by a fibrillary tremor, and in such cases inquiry should be made for thyroid enlargement, ocular prominence, cardio-vascular phenomena, and other neurasthenic symptoms, the tremor being really one of these.

Nervous Tremor arises from debility, and is seen in its most typical form in cases of *neurasthenia* and *general debility.* Tremor may also be present in *occupation neuroses.*

There are organic diseases in which tremors occur. *Tumour of the brain* may give rise to movements, especially (i.) when the tumour presses on, without actually destroying, some part of the Motor Tract. When the superior cerebellar peduncles are involved there may be tremor exactly like that of disseminated sclerosis. (ii.) If it be situated in the Optic Thalamus, the tremor usually takes the form of athetoid movements. (iii.) Cerebellar lesions may be associated with spasmodic movements of the neck muscles on one or other side. (iv.) Tumour in the Frontal Region may be attended by a fine tremor of the hand on the same side (T. Grainger Stewart). (v.) Lesions of the Red Nucleus or its connections with the Cerebellar Dentate nucleus of the opposite side or with the anterior horns below are attended by tremor.

Lateral sclerosis often gives rise to tremor of the legs on walking or any muscular movement.

Friedreich's disease is characterised by tremor or by disorderly movements.

§ 629. Chorea (St. Vitus' Dance, Sydenham's Chorea) is a disorder of the nervous system occurring generally in childhood, characterised by irregular awkward movements of the limbs and a tendency to cardiac valvular disease, running a more or less definite course usually towards

spontaneous recovery. It was first described by Sydenham in the seventeenth century. It is rheumatic in origin.

(1) The movements usually partake more of the character of gesticulations or exaggerations of normal movements than of tremors or even clonic spasms. In carrying a spoon to the mouth, the hand does not reach its goal, as it does in disseminated sclerosis. The movements may involve all the muscles of the body, even those of deglutition, the face, the tongue, and respiration, including the diaphragm. They are sometimes hemiplegic in distribution, and may predominate in the upper extremity or the face. The movements cease during sleep. The deep reflexes vary. (2) A certain amount of paresis may accompany or alternate with the movements, an t the chief symptom for which many patients are brought is their tendency to drop things or to fall down. Sometimes they are brought for restlessness at school. Very severe cases may develop maniacal symptoms. Eruptions on the skin (erythema, herpes zoster, purpura, and subcutaneous nodules) have been observed. (3) It is also accompanied, in a large proportion of recurrent cases, by valvular disease, which is indistinguishable from that of acute articular rheumatism. (4) There is a slight lymphocytosis in the cerebro-spinal fluid and increase of eosinophils in the blood.

The *Diagnosis* from habit spasm is occasionally difficult, but the latter is more persistent in duration, more limited in distribution, and the involuntary movements always affect the same set of muscles. It may also have to be diagnosed from various other clonic spasms mentioned below.

The *Prognosis* is usually favourable. The disease tends to spontaneous cure in the course of one to three months, though in about one-third of the cases permanent cardiac disease remains, and 20 per cent. of all cases get rheumatism within six years. The severity of the attack depends a good deal upon the age of the patient. Over the age of puberty chorea assumes a much graver aspect, and is very apt to be recurrent; and when it complicates pregnancy in young women the mortality is about 30 per cent. Violence of movement, frequent recurrence of attack, and maniacal symptoms (chorea insaniens) are always grave.

Etiology.—The disease is essentially one of childhood, and is three times more frequent in the female. There is a special liability for chorea to follow articular rheumatism, quinsy, scarlatina, and to a less extent other infective disorders. A family history of rheumatic fever is generally forthcoming. For many years I have taught that chorea is microbic in origin, pointing out in proof (1) the practical limitation of the disease to childhood; (2) its more or less definite course and tendency to spontaneous recovery; (3) the marked tendency to endocarditis. In 1903 Dr. J. F. Poynton revealed the rheumatic diplococcus in cases of chorea, and the disease is now coming to be regarded as microbic. As in rheumatism, there is a marked tendency to recurrence, and it is predisposed to by many of the same conditions as the acute specific fevers. Fright or other sudden emotion may determine an attack.

Treatment.--Salicylates as first introduced by Dr. David B. Lees, administered in the same way as in rheumatism, are almost as successful in chorea. Aspirin and quinine have been tried. The patients must be, taken from school, and put to bed, even the slight cases. The movements, if violent, may need bromides and large doses of chloral (10 grains every two hours if awake) or trional; chloretone is valuable. A water-bed is desirable. Arsenic though a popular remedy is valueless: large doses may check the movements by producing peripheral neuritis. A wet pack and other methods for the application of heat or cold are excellent means of treating severe cases (see § 633).

Huntingdon's Chorea (Chronic Chorea, Hereditary Chorea) characterised by irregular gesticulatory movements, coming on gradually between the thirtieth and fortieth year, and lasting for the rest of life. The gait is irregular and swaying, with sudden stoppages, the speech is affected, and there is mental impairment culminating in dementia. The disease runs in families. A similar affection coming on in the aged, without hereditary cause, is known as *senile chorea*. Rhythmical and other forms of chorea are mentioned below. It is unfortunate that the term "chorea" has been applied to these various conditions, which are totally distinct from Sydenham's chorea. The smaller cells and the corpus striatum are involved. *Henoch's chorea electrica* is probably identical with paramyoclonus multiplex (below).

Hysterical tremor and spasms are of different kinds. They may be generalised, like chorea, or localised to one situation; and they are aggravated by, but not dependent on, voluntary muscular action. They are characterised b(1) their sudden onset, generally after some emotional shock; (2) they vary in size, rhythm, and even in position from hour to hour and day to day; (3) they have a tendency to predominate in the head, neck, or arm; and (4) they occur in a characteristic age and sex. A class of hysterical spasm has been appropriately named chorée rhythmée by Charcot, in which condition the movements are distinctly choreiform, more or less generalised, and differ from ordinary chorea, first, by their coming on in attacks, or, at any rate, being liable to severe exacerbations; and, secondly, by having a tendency to a certain degree of regularity. In one case of this kind observed by me [1] these attacks could be started by pressure on the mamma, and they could be stopped by steady pressure in the ovarian region. In addition to these two forms, which might be called localised hysterical spasms and chorée rhythmée respectively, there are a large number of other rarer kinds, which may be provisionally placed in the hysterical group, though their precise relation to hysteria has not been made out. For instance, *saltatory spasm* (jumping chorea) is a rare condition of clonic spasms, affecting principally the lower extremities, which are usually in a state of more or less rigidity, and subject to violent extensor spasms whenever the soles of the feet are touched, or when the patient is placed upon the feet. The hands and arms are usually free. Hammering and dancing chorea have also been described by Charcot and others.

Myoclonus Multiplex (Synonym: Paramyoclonus Multiplex) is a very rare condition described by Friedreich.[2] It consists of attacks of sudden shock-like clonic spasms involving first one *single* muscle or *portion* of a muscle, and then another, not usually sufficiently prolonged to produce tremor of the limbs, bilateral, affecting mainly the proximal segments of the four extremities; it occurs mostly in males and runs a chronic course. Spasms of larger range may occur. A special variety associated with epilepsy has been described. It is probably due to some congenital or hereditary defect, though it may not be revealed for several years after birth. A most curious

[1] *Clinical Journal*, October 19, 1898.
[2] Virchow's Archiv, bd. lxxxvi., p. 421, 1881.

feature of this strange disorder is the exemption of the wrists, ankles, hands and feet. It is usually relieved by fairly strong galvanic currents, but is apt to relapse. The only case I have seen had lasted for nearly thirty years. Much confusion exists as to the identity of the disease.

The movements occurring in **Progressive Lenticular Degeneration** (§ 624) may in some cases resemble tonic or clonic spasms.

§ 630. Spasmodic Tic is a recurrent, involuntary, co-ordinated clonic spasm affecting certain groups of voluntary muscles, and producing twitchings which at first are limited to one region, but apt to spread. Many names have been applied to the condition—Habit Spasm, Convulsive Tic, Tic Nondouloureux, Impulsive Tic (Gilles de la Tourette), Spasmodic Torticollis— but they are essentially all the same. In the writer's view their pathology is similar, and depends on two factors : (a) An instability, natural or acquired, of the subconscious and reflex centres ; (b) a continued repetition of a certain movement, whereby it becomes exaggerated and in the end automatic. In the treatment of some of these cases the writer has been very successful with systematised muscular exercises and deep breathing.

Tic or **Habit Spasm** is a first cousin to chorea. It is, in fact, often difficult to distinguish between the two affections. It practically always starts in childhood, mostly in girls between seven and fourteen, and is, indeed, an exaggeration of the normal restlessness of that age. It consists of sudden quick muscular twitchings, say of the eyelide, face (causing grimaces), shoulder, or arm, differing from the chorea (i.) in being always limited to one region, (ii.) in the fact that exactly the same movements are repeated, and (iii.) in running a prolonged and indefinite course. The face and arms are the most frequent situations of the movements, but the muscles of respiration or any other part may be involved, and respiratory sniffs or grunts are not infrequent. It is always worst when the child is excited, or attempts to restrain the movements, and parents should be cautioned not to scold the child for the habit. There is frequently a history of neurosis in the family, and habit spasm may certainly arise by the "imitation" of other children. Medicines are not of much use. Removal from the surroundings under which the disease has arisen is the most efficient treatment.

A violent form of **Impulsive Tic** allied to habit spasm, and, like it, usually arising for the first time in children, though not confined to them, consists of explosive muscular movements, usually of the face or arms, but in violent cases of the entire body. Explosive sounds accompany these movements, either barking or inarticulate grunts, or the constant repetition of one word (echolalia), or some obscene or swearing word (coprolalia). The condition often occurs in association with some mental deficiency.

Localised Tonic Spasm may affect almost any voluntary muscle or group of muscles in the body. The case should be investigated on the lines below indicated under Facial Spasm. A large proportion are hysterical. In clonic spasm of the diaphragm curious respiratory spasms and grunts occur. Hysteria and gastric irritation may be causative factors here. It is also seen in organic cerebral disease. Clonic masticatory spasm may arise in paralysis agitans and old age, and sometimes in hysteria. Nutatory or nodding spasms occur in children at the time of dentition or as a form of epilepsy, sometimes associated with nystagmus.

§ 631. Clonic Facial Spasm (Synonyms : Spasmodic Tic, Convulsive Tic, Tic Nondouloureux, Mimic Spasm) is the term employed for a condition of persistent clonic spasm of the muscles of the face. Clonic facial spasm may arise under three different conditions : Organic lesion, reflex irritation, and idiopathic or constitutional causes. The favourite age for facial spasm is between thirty and sixty.

An *organic lesion* causing facial paralysis may give rise to post-paralytic facial

spasm. This is an indication either that the destruction of the facial nerve tracts was incomplete, or else that partial recovery has taken place (compare end of § 626). In this way tumours, such as sarcoma or aneurysm of the vertebral artery, pressing on without entirely destroying the facial nerve, give rise to facial spasm. Diseases of the bones (syphilitic or tuberculous) through which the nerve passes may also be suspected; and all the structures beside which the nerve passes should be carefully and thoroughly investigated. Strictly unilateral spasm involving all the muscles supplied by the facial, including the platysma and even the stapedius, without manifest cause, constitutes a distinct clinical variety. The seat of irritation in such cases must be either the facial nucleus or the nerve trunk. If the lesion be above the facial nucleus the spasm is more general, and is apt to involve muscles physiologically associated, perhaps on opposite sides, such as the anterior part of the occipito-frontalis. Thus, if both corrugators and the muscles of one cheek only be involved, a cortical lesion is indicated.

Reflex irritation, especially of the fifth nerve, may be in ·ration. The teeth should be examined, a history of injury to the fifth nerve it., ured for, pharyngeal adenoids removed, and the refraction should be tested.

Idiopathic Causes, such as grief or other emotion, debility, anæmia, the climacteric, hysteria, may act as contributory causes. It is this group that belongs to the class Spasmodic Tic (*vide supra*).

Facial spasm of *organic* origin may be *diagnosed* from habit spasm by its persistence, the age of the patient, the surrounding circumstances, and electrical changes (if any). But I know no means of distinguishing *non-organic* facial spasm from habit spasm in the face, though all the books describe them separately.

Course and Treatment.—Facial spasm, as above described, is generally a very chronic and progressive condition, resisting all our efforts excepting in those cases where the cause is removable, and the cases should be thoroughly investigated on the lines above given. Weak galvanism of the affected nerve may be tried. To palliate the spasm bromides, gelsemium, cimicifuga, antipyrin, chloral in extreme cases morphia and other nerve sedatives may be employed. When the spasm is due to a nuclear or nerve lesion, alcoholic injections administered through the stylo-mastoid foramen should be tried: the nerve is temporarily destroyed, and on regeneration tends to function normally.

§ 632. **Torticollis** (Wry-Neck) is a spasm of the muscles on one side of the neck. Wry-neck is of two kinds—the congenital, due, probably, to injury at birth: and the acquired, which may appear at any age. In the former condition the spasm is tonic ; in the latter the spasm is chiefly clonic, but in long-standing cases it tends to become tonic. (a) *Congenital* wry-neck is due to a contraction of the sterno-mastoid of one side, rarely both. The chin is projected upwards and to the opposite side. It may not be noticed by the parents till the child is several years old. It is generally associated with facial asymmetry, as pointed out by Sir Samuel Wilks. The only remedy is tenotomy. (b) *Acquired* torticollis consists of a slow clonic spasm recurring every few minutes, associated in long-standing cases with a certain amount of tonic spasm which, when the sterno-mastoid is involved (as is usual), draws the head to the opposite side. In about half the cases the trapezius is associated in the spasm ; in other cases the splenius, scalenus, and platysma may also be involved. The condition is very intractable.

Causes (compare also the remarks on Facial Spasm, which apply here) and *Treatment.*—The causes of torticollis are often obscure. The acquired form very commonly appears to be of hysterical origin. I have met with cases coming on after injury to the back of the head. Like facial spasm (*q.v.*), wry-neck probably arises under three different conditions—organic lesion, reflex irritation and idiopathically—and treatment should be directed to these. Many drugs have been tried, without much benefit. Galvanism of the spinal accessory nerve regularly applied twice daily has in one instance produced permanent relief. Surgical means (stretching, division or excision of the nerve, or division of the muscle and resection of the posterior branches of the upper

cervical nerves) have been tried; but as far as I am aware, without much success.

Muscular twitchings or startings may arise in muscles which are over-fatigued or during the half-waking state, in various conditions attended by constitutional debility -(q.v.). They are an occasional symptom of neurasthenia, and twitchings of the same kind, affecting the legs are also one of the earlier symptoms of peripheral neuritis.

§ 633. **Post-Paralytic Tremors and Spasms.**—The fact that hemiplegia and paraplegia of organic origin are gradually succeeded by tonic rigidity and other evidences of lateral sclerosis has already been referred to, but occasionally we also get clonic movements of various kinds supervening sooner or later, especially when the paralysis has occurred in early life.[1] There are many different kinds of post-paralytic clonic spasm, but practically they come under three types, which, in order of frequency, are (a) a slow mobile spasm called *athetosis* ; (b) movements which are more or less *rhythmical* ; and (c) irregular choreiform movements not inappropriately called *posthemiplegic chorea.* They are all characterised by (1) having the same distribution (generally hemiplegic, as that of the preceding paralysis, the arm in cases of hemiplegia being always more affected than the leg. (2) The presence of the other symptoms of lateral sclerosis. All of these movements are relatively rare after lesions occurring in adult life ; in such cases they are more frequent after hemiplegia due to an embolism than after hæmorrhage, and also after lesions which are associated with hemianæsthesia. The jerkings of the legs quite early in cases of paraplegia due to a complete transverse lesion with a fairly extensive area of healthy cord below, are caused, not by descending sclerosis, but rather by the cutting off of inhibitory influences from the brain.

The *Prognosis of Tremors and Clonic Spasms* in general terms is much more favourable than that of hemi- or para-plegia-- a fact which is in keeping with the remarks on pathology in § 626. The prognosis of some has been dealt with in detail. (1) In none of the above conditions is there, as a rule, any immediate danger to life, excepting in certain cases of chorea and in paralytic dementia. (2) There are but three of the above maladies which tend progressively to a lethal termination—viz., disseminated sclerosis, paralytic dementia, and progressive muscular atrophy. Disseminated sclerosis lasts on an average five or six years, being shortest in the cerebro-spinal form and longest in the spinal form, the cerebral form occupying an intermediate position. In all four of these the disease rarely lasts longer than ten years, and in acute cases only about a year or so. (3) In another group of the above diseases life is not materially shortened —viz., paralysis agitans, senile tremor, lateral sclerosis, many cases of facial and neck spasm, and some of the rarer group allied to hysteria. Nevertheless, some of these can be ameliorated. (4) Many of them progress towards spontaneous recovery—e.g., chorea, which is perhaps the most frequent clonic spasm met with, hysterical and nervous tremors, habit spasm. and many cases of facial spasm. (5) Many of the above are curable, chiefly by removing the cause—for example, alcoholic, metallic, and other toxic tremors, Graves' disease and the great majority of those which depend on reflex causes.

Indications for the Treatment of Muscular Tremor and Spasm.—For the rational treatment the reader should refer to §§ 626 and 630, giving the three pathological causes on which the cases may depend. The indications for treatment are fourfold. (1) To restore the partially damaged motor fibres. Iodide of potassium is of use, not only in cases of known syphilitic origin, to promote absorption, but also to eliminate toxic agents, such as lead, mercury, etc. (2) Careful investigation should always be made for any reflex irritation, such, for instance, as any uterine, gastric or other visceral trouble in cases of hysterical spasm. (3) The third indication is to remedy any constitutional or general defect, such as anæmia, rheumatism, debility, over-work, or over-worry. (4) If the cause cannot be ascertained or eradicated, we can, neverthe-

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[1] This is one of the facts supporting the belief that tremors and clonic spasms of organic origin are due to partial destruction or partial recovery of a motor nerve tract. It is the recuperative power of childhood which leads to partial recovery.

less, in many cases alleviate the tremor or spasm by appropriate means. Thus, hyoscyamus and its alkaloid (hyoscine hydrobromide, gr. ,¹₆₀ to ;¹₄ (0·0003 0·0012) ter die) are valuable in this respect, and many instances could be quoted of considerable relief from this drug, in cases of paralysis agitans,¹senile tremor, and disseminated sclerosis. Cannabis Indica, physostigma, conium (succus ʒss increased to ʒss (2–15) may also be tried. Opium and morphia are permissible in some severe cases, but, as a rule, the relief is only temporary. Bromides are undoubtedly of great value in neurasthenic and other nervous tremors, hysterical tremors and spasms, paralytic dementia, and chorea. Warm baths, Turkish baths, and the application of heat are agents for the relief of tremor and spasm which are too often neglected. Hot water internally and externally is the best remedy I know of for the relief of spasm of unstriped muscular fibres (such as spasmodic dysmenorrhœa); and the hot pack,¹ or hot bath, as mentioned before, acts wonderfully in cases of chorea. The very simplicity of this remedy –the application of heat—renders it all the more valuable. In cases where the paralysis is a leading feature strychnine may be given, though, as a rule, this remedy is contra-indicated in clonic spasms. Tremors and spasms of hysterical origin must be treated on general lines. Cold douches are useful for functional spasms. Localised muscular spasms are also treated by massage and electricity, the anodal pole being placed on the affected region.

The patient has **attacks** *of* **clonic and tonic spasms** *associated with more or less* DISTURBANCE OF CONSCIOUSNESS. The case is one of CONVULSIONS.

§ 634. **Convulsions** are sudden, violent, clonic, and sometimes tonic spasms affecting the greater part or the whole of the body, usually accompanied by some disturbance of consciousness.

The most common cause of convulsions is idiopathic epilepsy; convulsions arising from other causes may so closely resemble epilepsy that they are often described as epileptiform convulsions. The term eclampsia was formerly used for epileptiform convulsions. The following are the causes of convulsions :

Functional.

   I.   Idiopathic epilepsy.
  II.   Hysterical convulsions.

Organic Lesions.

 III.   Intracranial syphilis.
  IV.   Gross lesions of an irritative nature, such as (1) intracranial tumour (Jacksonian epilepsy); (2) hæmorrhage and embolism; (3) chronic degenerations of the nervous system; (4) acute meningitis.

Toxic Causes.

   V.   Uræmia, diabetes, cholæmia.
  VI.   Puerperal eclampsia.
 VII.   Various other toxæmic conditions, such as (1) alcohol; (2) lead; (3) drugs; (4) tetanus (tonic spasm), diabetes, malaria, acute specific fevers (in children).

Circulatory.

VIII.   Cardio-vascular disorders, including chronic Bright's disease.

Reflex.

  IX.   Reflex causes.

Convulsions in Infancy and Childhood.

¹ A thick blanket should be thoroughly wetted in a pail of boiling water, wrung out, and rolled up tight, and then the patient rolled in it, afterwards in another

The chief points in the CLINICAL INVESTIGATION of a convulsive attack are the age of the patient, the character of the attack, the condition immediately preceding and following it, and the attendant conditions. Ask *first* the *age*, and *secondly* whether the patient ever had an attack before. If the patient be under twelve months old, turn to Infantile Convulsions (§ 636). Between one and ten years of age embolism, hæmorrhage, and many constitutional derangements may give rise to convulsions. Between ten and twenty is the commonest time for idiopathic epilepsy to commence. If the patient be over thirty, and has never had a fit before, syphilis could be suspected. Hysterical convulsions affect chiefly the female sex between fifteen and twenty-five and at the climacteric. If the patient be over fifty, uræmia and apoplexy should be suspected. *Thirdly*, the *characters of the fit* afford considerable aid in diagnosis. Thus (i.) the convulsions of major epilepsy and major hysteria are always generalised; partial convulsions indicate usually a cortical lesion (Jacksonian epilepsy). (ii.) Unconsciousness is an invariable accompaniment of major epilepsy, and is very usual with uræmia, cerebral hæmorrhage, and syphilis. On the other hand, consciousness is not completely obliterated in by far the greater number of cases of hysterical convulsions, and in a considerable number of limited cortical lesions. *Fourthly*, inquiry should be made as to whether the fit was preceded by an aura (epilepsy), and what is the condition immediately afterwards. *Fifthly*, the history and attendant symptoms should be examined in the usual way. The presence of a blue line on the gums, or cutaneous syphilitic lesions, may decide the diagnosis of lead poisoning or syphilis. Lumbar puncture may be helpful in some cases.

§ 635. **Idiopathic Epilepsy** (Synonym : Falling Sickness) consists of sudden attacks of loss of consciousness, with or without convulsions, without any discoverable lesion. It occurs in two clinical forms, *minor epilepsy* (petit mal), which consists mainly of a transitory disturbance of consciousness, and is therefore referred to in § 562, and *major epilepsy* (haut mal, grand mal), which consists of a convulsive seizure with loss of consciousness.

Symptoms.—A complete epileptic fit has the following characters, though they are rarely all present in their entirety : (1) In some cases, during the previous twelve to twenty-four hours, there may be *prodromata*, consisting of headache, giddiness, malaise, or alteration of character or mood. In more than half the cases this stage is absent. (2) The fit in many cases is immediately preceded by an *aura* or warning—*i.e.*, a sensation or motion lasting at most only a few seconds, valuable as indicating the point of the cortex whence the cortical nerve-storm starts. The auræ differ infinitely in detail. There are four main groups. *Sensory auræ* are most common—*e.g.*, " a wave passing over the body," numbness, a sensation of movement, flashes of light or of colour, or singing in the ears ; *motor auræ*—*e.g.*, twitching of a muscle or a limb, occasionally of the trunk, and in some cases there is a " procursive aura," in which the patient runs forward or turns round and round ; *psychical auræ*—*e.g.*, various strange thoughts or hallucinations ; and, *somatic auræ*—*e.g.*, gastric discomfort, nausea, or fluttering in the stomach. Some form of aura is present, in my experience, in about three-quarters of the cases ; other physicians

dry up, and left for half an hour to perspire. It is often advisable to promote diaphoresis by a dose of liquor ammoniæ acetatis, for the benefit derivable is much less unless perspiration ensues.

give a smaller proportion. (3) *Loss of consciousness* is the pathognomonic and indispensable feature of idiopathic epilepsy, save the slighter forms of minor epilepsy *in which consciousness may be retained.* It succeeds the aura so quickly that the patient may not have time to place himself out of danger before loss of consciousness is complete. (4) *Convulsions* supervene almost at the same time as the unconsciousness. They are often ushered in with a scream, and in the classical form consist of a short stage of tonic convulsions lasting about forty seconds, followed by a stage of clonic convulsions lasting one to three minutes. In the tonic stage the . breath is held, the hands are clenched, the back is rigid, the legs are extended, the pulse is quick and may be imperceptible.° The clonic movements soon involve the whole body, and are sometimes of great violence, consisting of rapid extension and flexion of the limbs, opening and shutting of eyes and jaws. The interference of respiration during both the tonic and clonic stage causes an ever-increasing blueness of the face. The tongue is often bitten—a danger to avoid by thrusting a piece of wood, the handle of a pocket-knife, or something of the kind between the teeth. During both these stages the pupils are dilated and inactive to light, and the conjunctival reflex is absent. The light reflex may return during the . clonic stage. As the convulsions pass off the respiration becomes stertorous or snoring. Urine, fæces, and even semen may be voided. The saliva issues from the mouth as a frothy foam, sometimes blood-stained from injury of the tongue. (5) A stage of *stupor* or *drowsiness* succeeds the convulsions, gradually passing into a deep sleep. The temperature directly after the convulsions is said to be raised, sometimes as much as 4° or 5° F. (6) In the *post-epileptic state,* after recovery, there may be aphasia, or transient paresis, or the patient may perform automatic, irresponsible acts, dressing or undressing himself, or put the property of others into his own pocket, or he may pass into a state of somnambulism (dual personality) lasting for days together—or even longer. Occasionally hallucinations, delusions, or active mania ensue, or the patient makes obscene remarks or commits acts of violence. Some patients are distinctly homicidal, rarely suicidal. One fact of considerable forensic import must be mentioned—namely, the automatic phenomena of the post-epileptic state are by no means proportionate to the severity of the seizure (major or minor) which they follow—generally quite the reverse. Indeed the initial seizure may be so slight as to escape the notice of onlookers.

The *intervals* between the fits vary from a few days to many years. The patient usually remains in fairly good health of mind and body between the seizures ; indeed, he may profess himself in better health after an attack than before. *Epilepsy* has a close association with *insanity*. A certain proportion of cases, whether treated with bromides or not, drift into weakmindedness. Petit mal leads more often to mental defect than does epilepsy major. Epileptic fits may be followed (and sometimes even replaced) by mania of a dangerous nature, in which the patient may (quite unconsciously) commit acts of the most brutal kind. The affinity

between epilepsy and insanity is shown by heredity: one child may be epileptic, another insane.

Varieties.—The above description is of the classical form of epilepsy major, but every degree of severity is met with between this and epilepsy minor. Frequently one or the other, or several of the features enumerated, may be wanting, but unconsciousness is the one constant feature, except in some forms of epilepsy minor. Sometimes fits of various kinds may alternate in the same patient; but generally each patient has fits which conform to one, or at most to two, varieties. The *status epilepticus* is a rare condition in which the patient has a series of fits occurring in very rapid succession for several hours or even days, consciousness not being regained in the intervals; the temperature may rise to 107° F., and the issue is generally fatal.

The *Diagnosis* of epilepsy minor will be found in § 562. Epilepsy major may have to be diagnosed from any of the causes of convulsions (see list, § 634; see also table on following page). Convulsions coming on for the first time after thirty are more probably *syphilitic* than idiopathic. In *feigned epilepsy* the pupils are not dilated, and they react normally to light, the conjunctivæ are sensitive, and the application of strong ammonia to the nostrils generally reveals the fraud; the absence of cyanosis may also aid. The alleged fact that the impostor will not hurt himself must not too readily be relied upon: he may be prepared to do himself injury if the motive for the fraud is sufficiently powerful.

Course and Prognosis.—If unchecked by treatment the fits tend to recur throughout life, though with widely varying frequency. The frequency of the fits is the leading factor in the prognosis. Those in whom they occur with moderate severity a few times a year may remain well and clever, but when more frequent, mental deterioration results, not, be it remembered, as a consequence of the administration of bromide, but as a part of the disease. Indeed, in petit mal, on which bromide has least influence, mental symptoms ensue more often than in grand mal. A combination of the two forms is worse than either singly. The earlier the fits commence, and the stronger the heredity factor, the worse the prognosis. Death may occur from an accident during the fit, but rarely from the disease.

Etiology.—Both sexes are about equally affected. In about 75 per cent. the fits start before the age of fourteen; in only a small percentage do they begin after twenty. Heredity is a potent factor. Among the determining causes of a fit may be mentioned fright, excitement, head injuries, the menstrual period (many females have fits only at this time), alcoholic and other excesses. Epileptic fits have been known to be associated reflexly with such causes as the irritation of a scar, disease of the eyeball, disease of the nasal mucous membrane, on the removal of which the fits have ceased. It has been shown, moreover, that in a small proportion of epileptics benefit may result from the correction of an error of refraction.

TABLE OF DIAGNOSIS OF EPILEPSY.

	Major Epilepsy.	Hysterical Convulsions.	Feigned Epilepsy.
Preceded by	Characteristic aura frequent.	Globus, or choking, epigastric sinking or emotional disturbance.	A motive for fraud.
Mode of onset.	Sudden; sometimes with one typical cry.	Sometimes gradual. perhaps with screams.	Carefully planned.
Characters of convulsions.	March definite and noiseless. Tonic brief, followed by clonic movements, which are wholly non-purposive ("inco-ordinated"). Cyanosis during tonic stage; tongue may be bitten; urine and faeces may be voided. Often injured in the fall.	Progress irregular, with screaming or cry. Tonic rigidity often prolonged and recurrent. Clonic movements often purposive or semi-purposive; tongue not bitten; urine and faeces rarely voided; usually no cyanosis. Rarely injured.	Not following the usual march; absence of cyanosis and asphyxia; urine and faeces not voided; tongue not bitten.
Consciousness.	Always lost.	Never quite lost.	Retained.
Eye symptoms.	Pupils dilated and irresponsive to light; conjunctivæ insensitive.	Pupils responsive to light; conjunctivæ sensitive.	Conjunctiva sensitive; pupils responsive to light.
Duration.	Rarely exceeds a few minutes.	Generally exceeds 4 or 5 minutes, may last half an hour and liable to recur.	Indefinite.
Termination.	Stupor and drowsiness.	Sobbing, crying, laughter, or prostration.	Not followed by profound stupor.

Hughlings Jackson believed that every epileptic patient has an epileptogenic zone or area on the surface of the body corresponding to the region in the brain whence the cortical discharge starts; but in actual practice it is rarely possible to discover such a zone.

Three cases have been recorded in which fits, similar to those from which the patient suffered spontaneously, could be induced by a slight cutaneous irritation in a definite area. In Dr. Jackson's case, a boy aged eight, a flick with a silk handkerchief on the back of the head from behind, so that he received no warning, would result in his immediately falling down in a fit. In 1897, at the age of nineteen, this boy was under my care, and he presented hemiplegia and unmistakable signs of descending sclerosis and commencing athetosis. It is an interesting fact that all three presented certain features in common. All were children—two boys and one girl—and in all the fits began early in life. In all the epileptogenic zone was situated in the head, and it was only when the patients were unaware that they were going to be touched that the fits were produced. In falling, it was noticed that these patients generally struck their heads with great violence, suggesting that the clonic convulsions began in the neck muscles, pulling down the head in one direction. There were evidences in all pointing to a gross lesion of the brain as a cause of the fits. The two boys were hemiplegic, and the hemiplegia was increased after the fits. The girl was not hemiplegic, but in her the history of a series of convulsions at about three, followed by two years' interval, points in the same direction (see § 000 below).

Treatment.—Luminal gr. 1½ once or twice daily, and bromides 20–30